

WEST Search History

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DATE: Monday, January 17, 2005

Hide?	Set Name	Query	Hit Count
		<i>DB=USPT; PLUR=YES; OP=OR</i>	
<input type="checkbox"/>	L7	L6 same (IL-12 or interleukin-12)	14
<input type="checkbox"/>	L6	(inhibit or scavenge) with (NO or nitric adj oxide).	15878
		<i>DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI; PLUR=YES; OP=OR</i>	
<input type="checkbox"/>	L5	L4 and (IL-12 or interleukin-12)	45
<input type="checkbox"/>	L4	monomethyl adj arginine or (l-NMMA)	424
		<i>DB=USPT; PLUR=YES; OP=OR</i>	
<input type="checkbox"/>	L3	L2 and (IL-12 or interleukin-12)	4
<input type="checkbox"/>	L2	monomethyl adj arginine	63
<input type="checkbox"/>	L1	monomethyl adj arginine with inhibitor	19

END OF SEARCH HISTORY

FILE 'CAPLUS' ENTERED AT 18:21:44 ON 17 JAN 2005
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
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FILE 'MEDLINE' ENTERED AT 18:21:44 ON 17 JAN 2005

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FILE 'USPATFULL' ENTERED AT 18:21:44 ON 17 JAN 2005
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FILE 'BIOTECHNO' ENTERED AT 18:21:44 ON 17 JAN 2005
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=> s (IL-12 or interleukin-12) (a) (nitric (w) oxide (w) inhibit or nitric (w) oxide (w) neutraliz?)
L1 0 (IL-12 OR INTERLEUKIN-12) (A)
(NITRIC (W) OXIDE (W) INHIBIT OR
NITRIC (W) OXIDE (W) NEUTRALIZ?)

=> s (IL-12 or interleukin-12) and (nitric (w) oxide (w) inhibit or nitric (w) oxide (w) neutraliz?)
L2 3 (IL-12 OR INTERLEUKIN-12) AND
(NITRIC (W) OXIDE (W) INHIBIT OR
NITRIC (W) OXIDE (W) NEUTRALIZ?)

=> duplicare remove l2
DUPLICARE IS NOT A RECOGNIZED
COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> duplicate remove l2
DUPLICATE PREFERENCE IS 'CAPLUS',
USPATFULL'
KEEP DUPLICATES FROM MORE THAN ONE
FILE? Y/(N):n
PROCESSING COMPLETED FOR L2
L3 3 DUPLICATE REMOVE L2 (0
DUPLICATES REMOVED)

=> d l2ibib, abs
'L2IBIB' IS NOT A VALID FORMAT
In a multifile environment, a format can only be used if it is valid
in at least one of the files. Refer to file specific help messages
or the STNGUIDE file for information on formats available in individual files.

REENTER DISPLAY FORMAT FOR ALL FILES
(FILEDEFAULT):l2 ibib abs
'L14' IS NOT A VALID FORMAT

In a multifile environment, a format can only be used if it is valid
in at least one of the files. Refer to file specific help messages
or the STNGUIDE file for information on formats available in individual files.

REENTER DISPLAY FORMAT FOR ALL FILES
(FILEDEFAULT):d l2 ibib abs
'D' IS NOT A VALID FORMAT

'L14' IS NOT A VALID FORMAT
In a multifile environment, a format can only be used if it is valid
in at least one of the files. Refer to file specific help messages
or the STNGUIDE file for information on formats available in individual files.

REENTER DISPLAY FORMAT FOR ALL FILES
(FILEDEFAULT):l2 ibib abs
'L14' IS NOT A VALID FORMAT

In a multifile environment, a format can only be used if it is valid
in at least one of the files. Refer to file specific help messages
or the STNGUIDE file for information on formats available in individual files.

REENTER DISPLAY FORMAT FOR ALL FILES
(FILEDEFAULT): d ibib abs
'D' IS NOT A VALID FORMAT

In a multifile environment, a format can only be used if it is valid
in at least one of the files. Refer to file specific help messages
or the STNGUIDE file for information on formats available in individual files.
REENTER DISPLAY FORMAT FOR ALL FILES
(FILEDEFAULT):ibib abs

L3 ANSWER 1 OF 3 USPATFULL on STN
ACCESSION NUMBER: 2002:156684
USPATFULL
TITLE: Methods and compositions for
enhancing the

immunostimulatory effect of
interleukin -
12

INVENTOR(S): Trinchieri, Giorgio, Charly,
FRANCE
Lee, William M. F., Wynnewood, PA,
UNITED STATES
Koblish, Holly, Yardley, PA, UNITED
STATES

NUMBER KIND DATE

PATENT INFORMATION: US 2002081277 A1
20020627
APPLICATION INFO.: US 2002-79068 A1
20020220 (10)
RELATED APPLN. INFO.: Division of Ser. No. US
1999-395038, filed on 13 Sep
1999, GRANTED, Pat. No. US
6375944

NUMBER DATE

PRIORITY INFORMATION: US 1998-101698P
19980925 (60)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: HOWSON AND
HOWSON, ONE SPRING HOUSE CORPORATION
CENTER,
BOX 457, 321 NORRISTOWN
ROAD, SPRING HOUSE, PA, 19477
NUMBER OF CLAIMS: 24
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 7 Drawing Page(s)
LINE COUNT: 1155
CAS INDEXING IS AVAILABLE FOR THIS
PATENT.
AB Methods for enhancing the therapeutic and
adjuvant use of ***IL*** -
12 by reducing unwanted transient
immunosuppression caused by
IL - ***12*** or by high doses thereof
involve
co-administering ***IL*** - ***12*** with
an effective amount of
an agent that inhibits or neutralizes nitric oxide
(NO) in vivo.
Enhanced vaccine therapy involves co-
administering the ***IL*** -
12 adjuvant, a selected vaccine antigen
and the NO
inhibiting/neutralizing agent. Additionally, the
toxicity of ***IL***
- ***12*** treatment may be reduced by co-
administering ***IL*** -
12 with an effective amount of the NO
inhibiting or neutralizing
agent. A therapeutic composition characterized by
reduced toxicity in
mammals contains ***IL*** - ***12*** ,
preferably a low dose
thereof, and an NO inhibiting or neutralizing
agent in a

pharmaceutically acceptable carrier. A vaccine
composition contains an
effective adjuvanting amount of ***IL*** -
12 , an effective
amount of an NO inhibiting or neutralizing agent,
and an effective
protective amount of a vaccine antigen in a
pharmaceutically acceptable
carrier.

CAS INDEXING IS AVAILABLE FOR THIS
PATENT.

=> d 1- ibib abs
YOU HAVE REQUESTED DATA FROM 3
ANSWERS - CONTINUE? Y/(N):y

L3 ANSWER 1 OF 3 USPATFULL on STN
ACCESSION NUMBER: 2002:156684
USPATFULL
TITLE: Methods and compositions for
enhancing the
immunostimulatory effect of
interleukin -
12
INVENTOR(S): Trinchieri, Giorgio, Charly,
FRANCE
Lee, William M. F., Wynnewood, PA,
UNITED STATES
Koblish, Holly, Yardley, PA, UNITED
STATES

NUMBER KIND DATE

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1999-395038, filed on 13 Sep
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LEGAL REPRESENTATIVE: HOWSON AND
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BOX 457, 321 NORRISTOWN
ROAD, SPRING HOUSE, PA, 19477
NUMBER OF CLAIMS: 24
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 7 Drawing Page(s)
LINE COUNT: 1155
CAS INDEXING IS AVAILABLE FOR THIS
PATENT.

AB Methods for enhancing the therapeutic and adjuvant use of ***IL*** -
 12 by reducing unwanted transient immunosuppression caused by
 IL - ***12*** or by high doses thereof involve
 co-administering ***IL*** - ***12*** with an effective amount of
 an agent that inhibits or neutralizes nitric oxide (NO) in vivo.
 Enhanced vaccine therapy involves co-administering the ***IL*** -
 12 adjuvant, a selected vaccine antigen and the NO
 inhibiting/neutralizing agent. Additionally, the toxicity of ***IL***
 - ***12*** treatment may be reduced by co-administering ***IL*** -
 12 with an effective amount of the NO inhibiting or neutralizing
 agent. A therapeutic composition characterized by reduced toxicity in
 mammals contains ***IL*** - ***12*** , preferably a low dose
 thereof, and an NO inhibiting or neutralizing agent in a
 pharmaceutically acceptable carrier. A vaccine composition contains an
 effective adjuvanting amount of ***IL*** - ***12*** , an effective
 amount of an NO inhibiting or neutralizing agent, and an effective
 protective amount of a vaccine antigen in a pharmaceutically acceptable
 carrier.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 2 OF 3 USPATFULL on STN
 ACCESSION NUMBER: 2002:87988
 USPATFULL

TITLE: Methods and compositions for enhancing the

immunostimulatory effect of
 interleukin -
 12

INVENTOR(S): Trinchieri, Giorgio, Charly,
 FRANCE

Lee, William M. F., Wynnewood, PA,
 United States

Koblish, Holly, Yardley, PA, United
 States

PATENT ASSIGNEE(S): The Wistar Institute of
 Anatomy and Biology,

Philadelphia, PA, United States (U.S.
 corporation)

The Trustees of the University of
 Pennsylvania,
 Philadelphia, PA, United States (U.S.
 corporation)

NUMBER KIND DATE

PATENT INFORMATION: US 6375944 B1
 20020423
 APPLICATION INFO.: US 1999-395038
 19990913 (9)

NUMBER DATE

PRIORITY INFORMATION: US 1998-101698P
 19980925 (60)
 DOCUMENT TYPE: Utility
 FILE SEGMENT: GRANTED
 PRIMARY EXAMINER: Mertz, Prema
 ASSISTANT EXAMINER: Prasad, Sarada C
 LEGAL REPRESENTATIVE: Howson and Howson
 NUMBER OF CLAIMS: 18
 EXEMPLARY CLAIM: 1
 NUMBER OF DRAWINGS: 10 Drawing Figure(s);
 7 Drawing Page(s)
 LINE COUNT: 1207
 CAS INDEXING IS AVAILABLE FOR THIS
 PATENT.

AB Methods for enhancing the therapeutic and adjuvant use of ***IL*** -
 12 by reducing unwanted transient immunosuppression caused by
 IL - ***12*** or by high doses thereof involve
 co-administering ***IL*** - ***12*** with an effective amount of
 an agent that inhibits or neutralizes nitric oxide (NO) in vivo.
 Enhanced vaccine therapy involves co-administering the ***IL*** -
 12 adjuvant, a selected vaccine antigen and the NO
 inhibiting/neutralizing agent. Additionally, the toxicity of ***IL***
 - ***12*** treatment may be reduced by co-administering ***IL*** -
 12 with an effective amount of the NO inhibiting or neutralizing
 agent. A therapeutic composition characterized by reduced toxicity in
 mammals contains ***IL*** - ***12*** , preferably a low dose
 thereof, and an NO inhibiting or neutralizing agent in a
 pharmaceutically acceptable carrier. A vaccine composition contains an
 effective adjuvanting amount of ***IL*** - ***12*** , an effective
 amount of an NO inhibiting or neutralizing agent, and an effective
 protective amount of a vaccine antigen in a pharmaceutically acceptable
 carrier.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2005

ACS on STN

ACCESSION NUMBER: 2000:60896 CAPLUS

TITLE: Papers to Appear in Forthcoming
Issues

AUTHOR(S): Anon.

SOURCE: Cellular Immunology (1999),
198(2), 144

CODEN: CLIMB8; ISSN: 0008-8749

PUBLISHER: Academic Press

DOCUMENT TYPE: Journal; Miscellaneous

LANGUAGE: English

AB Human Leptin Enhances Activation and
Proliferation of Human Circulating T
Lymphocytes. Consuelo Martin-Romero, Jose
Santos-Alvarez, Raimundo

Goberna, and Victor Sanchez-Margalet. (Received
6/17/99; accepted

11/24/99.)Induction of ***Interleukin*** -
12 /p40 by

Superantigens in Macrophages Is Mediated by
Activation of Nuclear

Factor-.kappa.B. Caigan Du and Subramaniam
Sriram. (Received 9/16/99;

accepted 11/29/99.)alpha.6.beta.1 Integrin (VLA-
6) Mediates Leukocyte

Tether and Arrest on Laminin under Physiol. Shear
Flow. Joji Kitayama,

Shigeo Ikeda, Kyoko Kumagai, Hideaki Saito, and
Hirokazu Nagawa. (Received

8/26/99; accepted 12/1/99.)Macrophage-Derived
Nitric

Oxide ***Inhibits*** the Proliferation
of Activated T Helper

Cells and Is Induced during Antigenic Stimulation
of Resting T Cells.

Roel C. van der Veen, Therese A. Dietlin, J. Dixon
Gray, and Wendy

Gilmore. (Received 8/26/99; accepted
12/1/99.)Synthetic Melanin Suppresses

Prodn. of Proinflammatory Cytokines. Nahid
Mohagheghpour, Nahid Waleh,

Stephen J. Garger, Linda Dousman, Laurence K.
Grill, and Daniel Tuse.

(Received 7/12/99; accepted 12/6/99.)Cell-Specific
Inhibition of Inducible

Nitric Oxide Synthase Activation by Leflunomide.
V. Jankovic, T.

Samardzic, S. Stosic-Grujicic, D. Popadic, and V.
Trajkovic. (Received

7/13/99; accepted 12/6/99.)Regulation of Human
Natural Killer Cell

Migration and Proliferation by the Exodus
Subfamily of CC Chemokines.

Michael J. Robertson, Brian T. Williams, Kent
Christopherson II, Zacharie

Brahmi, and Robert Hromas. (Received 4/27/99;
accepted

12/7/99.)alpha.-Galactosylceramide Induces Early
B-Cell Activation

through IL-4 Prodn. by NKT Cells. Hidemitsu
Kitamura, Akio Ohta, Masashi

Sekimoto, Marimo Sato, Kenji Iwakabe, Minoru
Nakui, Takashi Yahata, Hongxu

Meng, Toshiaki Koda, Shin-ichiro Nishimura,
Tetsu Kawano, Masaru

Taniguchi, and Takashi Nishimura. (Received
7/26/99; accepted 12/9/99.).

(c) 1999 Academic Press.

=> s (IL-12 or interleukin-12) and (NO (w) inhibit? or
NO (w) neutraliz?)

5 FILES SEARCHED...

L4 164 (IL-12 OR INTERLEUKIN-12) AND
(NO (W) INHIBIT? OR NO (W) NEUTRAL
IZ?)

=> duplicate remove l4

DUPLICATE PREFERENCE IS 'CAPLUS,
MEDLINE, BIOSIS, EMBASE, USPATFULL,
BIOTECHNO'

KEEP DUPLICATES FROM MORE THAN ONE
FILE? Y/(N):n

PROCESSING COMPLETED FOR L4

L5 74 DUPLICATE REMOVE L4 (90
DUPLICATES REMOVED)

=> d 1- ibib abs

YOU HAVE REQUESTED DATA FROM 74
ANSWERS - CONTINUE? Y/(N):y

L5 ANSWER 1 OF 74 USPATFULL on STN

ACCESSION NUMBER: 2005:10995

USPATFULL

TITLE: Methods of screening for a
candidate modulator of
glucokinase

INVENTOR(S): Rizzo, Mark A., Nashville,
TN, UNITED STATES

Piston, David W., Nashville, TN,
UNITED STATES

NUMBER KIND DATE

PATENT INFORMATION: US 2005009129 A1
20050113

APPLICATION INFO.: US 2004-838167 A1
20040503 (10)

NUMBER DATE

PRIORITY INFORMATION: US 2003-467885P
20030505 (60)

DOCUMENT TYPE: Utility

FILE SEGMENT: APPLICATION

LEGAL REPRESENTATIVE: Steven L. Highlander,
FULBRIGHT & JAWORSKI L.L.P., 600

Congress Avenue, Suite 2400, Austin,
TX, 78701

NUMBER OF CLAIMS: 49

EXEMPLARY CLAIM: 1

NUMBER OF DRAWINGS: 6 Drawing Page(s)

LINE COUNT: 1679

AB The present invention relates to providing novel therapeutics for treating diabetes other glycemic disorders. Such therapeutics involve the signaling pathways that contribute to regulation of glucose-stimulated insulin secretion. Of particular interest are modulators of a key component in the glucokinase pathway. Thus, the present provides methods of screening for modulators of glucokinase activity, expression, translocation, conformation, nitrosylation and interaction with other molecules as useful target for pharmacological manipulation in the treatment of diabetes and other glycemic disorders.

L5 ANSWER 2 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2004:326849
USPATFULL

TITLE: Method for regulating the expression of genes carried on a viral vector

INVENTOR(S): Inoue, Makoto, Ibaraki, JAPAN

Iida, Akihiro, Ibaraki, JAPAN
Hasegawa, Mamoru, Ibaraki, JAPAN

NUMBER KIND DATE

PATENT INFORMATION: US 2004258668 A1
20041223
APPLICATION INFO.: US 2004-489394 A1
20040813 (10)

WO 2002-JP10065 20020927

NUMBER DATE

PRIORITY INFORMATION: JP 2001-298223
20010927

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: CLARK & ELBING
LLP, 101 FEDERAL STREET, BOSTON, MA,
02110

NUMBER OF CLAIMS: 11
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 13 Drawing Page(s)
LINE COUNT: 810

CAS INDEXING IS AVAILABLE FOR THIS
PATENT.

AB The present inventors revealed that non-steroidal anti-inflammatory drugs and muscle relaxants suppressed the expression of genes carried on viral vectors. These agents also suppressed viral vector cytotoxicity.

The present invention provides a method for regulating the expression of

genes carried on viruses by using non-steroidal anti-inflammatory drugs and/or muscle relaxants. The effect of these agents is reversible, and viral vector gene expression and cytotoxicity increased after termination of agent administration. The agents of the present invention are useful for regulating the expression of viral and therapeutic genes, and for suppressing viral vector cytotoxicity in gene therapy using viral vectors.

CAS INDEXING IS AVAILABLE FOR THIS
PATENT.

L5 ANSWER 3 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2004:313900
USPATFULL

TITLE: Auditory nerve protection and re-growth

INVENTOR(S): Miller, Josef M., Ann Arbor, MI, UNITED STATES

Altschuler, Richard A., Ann Arbor, MI,
UNITED STATES

Raphael, Yehoash, Ann Arbor, MI,
UNITED STATES

NUMBER KIND DATE

PATENT INFORMATION: US 2004247570 A1
20041209
APPLICATION INFO.: US 2003-345731 A1
20030116 (10)

NUMBER DATE

PRIORITY INFORMATION: US 2002-349799P
20020117 (60)

US 2002-351870P 20020125 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Tanya A Arenson,
MEDLEN & CARROLL LLP, Suite 350, 101
Howard Street, San Francisco, CA,

94105

NUMBER OF CLAIMS: 24
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 21 Drawing Page(s)
LINE COUNT: 2235

AB The present invention relates to compositions and methods for the protection and restoration of hearing. In particular, the present invention relates to treatments to facilitate the protection and re-growth of the auditory nerve. The present invention further provides methods of preventing hair cell loss and the accompanying loss in

hearing. The present invention thus provides novel interventions for a variety of hearing impairments.

L5 ANSWER 4 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2004:306481
USPATFULL
TITLE: Use of unmethylatd cpg
INVENTOR(S): De Simone, Claudio, Ardea
Rm, ITALY

NUMBER KIND DATE

PATENT INFORMATION: US 2004241149 A1
20041202
APPLICATION INFO.: US 2004-488606 A1
20040303 (10)
WO 2002-IT534 20020809

NUMBER DATE

PRIORITY INFORMATION: US 2001-316953P
20010905 (60)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: NIXON &
VANDERHYE, PC, 1100 N GLEBE ROAD, 8TH
FLOOR,

ARLINGTON, VA, 22201-4714

NUMBER OF CLAIMS: 28
EXEMPLARY CLAIM: 1
LINE COUNT: 897
CAS INDEXING IS AVAILABLE FOR THIS
PATENT.
AB Lactic acid bacteria containing unmethylated
cytosine-guanine (CpG)
dinucleotide are used to positively affect the
immune response in a
subject having or at risk of having an
inflammatory response to
lipopolysaccharides.

CAS INDEXING IS AVAILABLE FOR THIS
PATENT.

L5 ANSWER 5 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2004:281091
USPATFULL
TITLE: Compositions for, and methods of,
treating

atherosclerosis

INVENTOR(S): Romanczyk,, Leo J., JR.,
Hackettstown, NJ, UNITED
STATES
Schmitz, Harold H., Branchburg, NJ,
UNITED STATES

NUMBER KIND DATE

PATENT INFORMATION: US 2004220392 A1
20041104

APPLICATION INFO.: US 2004-770969 A1
20040506 (10)
RELATED APPLN. INFO.: Division of Ser. No. US
2002-127817, filed on 22 Apr
2002, PENDING Continuation of Ser.
No. US 2001-776649,
filed on 5 Feb 2001, GRANTED, Pat.
No. US 6638971
Continuation of Ser. No. US 1997-
831245, filed on 2 Apr
1997, GRANTED, Pat. No. US
6297273 Continuation-in-part
of Ser. No. US 1996-631661, filed on 2
Apr 1996,
ABANDONED Continuation of Ser.
No. US 2000-717893,
filed on 21 Nov 2000, GRANTED, Pat.
No. US 6670390
Continuation of Ser. No. US 1997-
831245, filed on 2 Apr
1997, GRANTED, Pat. No. US
6297273 Continuation-in-part
of Ser. No. US 1996-631661, filed on 2
Apr 1996,
ABANDONED
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: NADA JAIN, P.C.,
560 White Plains Road, Suite 460,
Tarrytown, NY, 10591
NUMBER OF CLAIMS: 17
EXEMPLARY CLAIM: CLM-1-208
NUMBER OF DRAWINGS: 242 Drawing Page(s)
LINE COUNT: 4732
CAS INDEXING IS AVAILABLE FOR THIS
PATENT.
AB Disclosed and claimed are cocoa extracts,
compounds, combinations
thereof and compositions containing the same,
such as polyphenols or
procyanidins, methods for preparing such extracts,
compounds and
compositions, as well as uses for them, especially
a polymeric compound
of the formula A.sub.n, wherein A is a monomer
of the formula:
##STR1##
wherein n is an integer from 2 to 18, such that
there is at least one
terminal monomeric unit A, and one or a plurality
of additional
monomeric units;
R is 3-(.alpha.)-OH, 3-(.beta.)-OH, 3-(.alpha.)-o-
sugar, or
3-(.beta.)-O-sugar;
bonding between adjacent monomers takes place
at positions 4, 6 or 8;

a bond of an additional monomeric unit in position 4 has alpha or beta stereochemistry;

X, Y and Z are selected from the group consisting of monomeric unit A, hydrogen, and a sugar, with the provisos that as to the at least one terminal monomeric unit, bonding of the additional monomeric unit thereto (the bonding of the additional monomeric unit adjacent to the terminal monomeric unit) is at position 4 and optionally Y=Z=hydrogen;

the sugar is optionally substituted with a phenolic moiety, at any position on the sugar, for instance via an ester bond, and

pharmaceutically acceptable salts or derivatives thereof (including oxidation products).

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 6 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2004:69535
USPATFULL
TITLE: Stat3 agonists and antagonists and therapeutic uses thereof
INVENTOR(S): Yu, Hua, Tampa, FL, UNITED STATES
Pardoll, Drew, Brookeville, MD, UNITED STATES
Jove, Richard, Tampa, FL, UNITED STATES
Dalton, William, Tampa, FL, UNITED STATES

NUMBER	KIND	DATE
PATENT INFORMATION: US 2004052762 A1 20040318		
APPLICATION INFO.: US 2003-380020 A1 20030902 (10)		
WO 2001-US28254 20010910		
DOCUMENT TYPE: Utility		
FILE SEGMENT: APPLICATION		
LEGAL REPRESENTATIVE: JONES DAY, 222 EAST 41ST STREET, NEW YORK, NY, 10017		
NUMBER OF CLAIMS: 49		
EXEMPLARY CLAIM: 1		
NUMBER OF DRAWINGS: 13 Drawing Page(s)		
LINE COUNT: 3467		
CAS INDEXING IS AVAILABLE FOR THIS PATENT.		

AB The present invention relates to methods for modulating, i.e., agonizing

or antagonizing, Stat3 (Signal Transducer and Activator of Transcription3) signaling activity for use in gene therapy. Inhibition and/or activation of Stat3 signaling is an effective approach to modulate angiogenesis and the immune response for treatment and/or prevention of inflammation, infection, inflammation, immune disorders, and ischemia.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 7 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2004:57395
USPATFULL
TITLE: IL-17 homologous polypeptides and therapeutic uses thereof
INVENTOR(S): Chen, Jian, Princeton, NJ, UNITED STATES
Filvaroff, Ellen, San Francisco, CA, UNITED STATES
Fong, Sherman, Alameda, CA, UNITED STATES
Goddard, Audrey, San Francisco, CA, UNITED STATES
Godowski, Paul, Hillsborough, CA, UNITED STATES
Grimaldi, Christopher, San Francisco, CA, UNITED STATES
Gurney, Austin, Belmont, CA, UNITED STATES
Li, Hanzhong, San Mateo, CA, UNITED STATES
Hillan, Kenneth, San Francisco, CA, UNITED STATES
Tumas, Daniel, Orinda, CA, UNITED STATES
VanLookeren, Menno, San Francisco, CA, UNITED STATES
Vandlen, Richard, Hillsborough, CA, UNITED STATES
Watanabe, Colin K., Moraga, CA, UNITED STATES
Williams, P. Mickey, Half Moon Bay, CA, UNITED STATES
Wood, William I., Hillsborough, CA, UNITED STATES
Yansura, Daniel, Pacifica, CA, UNITED STATES
PATENT ASSIGNEE(S): Genentech, Inc., South San Francisco, CA (U.S. corporation)

NUMBER	KIND	DATE
PATENT INFORMATION: US 2004043397 A1 20040304		

APPLICATION INFO.: US 2003-408385 A1
20030407 (10)
RELATED APPLN. INFO.: Division of Ser. No. US
2000-747259, filed on 20 Dec
2000, GRANTED, Pat. No. US
6569645 Continuation-in-part
of Ser. No. WO 2000-US30873, filed
on 10 Nov 2000,
PENDING Continuation-in-part of Ser.
No. WO
2000-US23328, filed on 24 Aug 2000,
PENDING

NUMBER	DATE

PRIORITY INFORMATION: US 2000-175481P	
20000111 (60)	
DOCUMENT TYPE:	Utility
FILE SEGMENT:	APPLICATION
LEGAL REPRESENTATIVE: GENENTECH, INC.,	
1 DNA WAY, SOUTH SAN FRANCISCO, CA,	
94080	
NUMBER OF CLAIMS:	60
EXEMPLARY CLAIM:	1
NUMBER OF DRAWINGS:	46 Drawing Page(s)
LINE COUNT:	8591
CAS INDEXING IS AVAILABLE FOR THIS	
PATENT.	
AB The present invention is directed to novel	
polypeptides and to nucleic	
acid molecules encoding those polypeptides. Also	
provided herein are	
vectors and host cells comprising those nucleic	
acid sequences, chimeric	
polypeptide molecules comprising the	
polypeptides of the present	
invention fused to heterologous polypeptide	
sequences, antibodies which	
bind to the polypeptides of the present invention	
and to methods for	
producing the polypeptides of the present	
invention.	

CAS INDEXING IS AVAILABLE FOR THIS
PATENT.

L5 ANSWER 8 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2004:46811
USPATFULL
TITLE: Procyanidin and cyclo-oxygenase
modulator compositions
INVENTOR(S): Romanczyk, Jr., Leo J.,
Hackettstown, NJ, United States
Schmitz, Harold H., Branchburg, NJ,
United States
PATENT ASSIGNEE(S): Mars, Incorporated,
McLean, VA, United States (U.S.
corporation)

NUMBER	KIND	DATE

PATENT INFORMATION: US 6696485 B1
20040224
APPLICATION INFO.: US 2002-268718
20021010 (10)
RELATED APPLN. INFO.: Continuation of Ser. No.
US 2000-717893, filed on 21
Nov 2000 Continuation of Ser. No. US
2001-776649, filed
on 5 Feb 2001 Continuation of Ser. No.
US 2002-127817,
filed on 22 Apr 2002
DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Solola, Taofiq
LEGAL REPRESENTATIVE: Nada Jain, P.C., Jain,
Nada
NUMBER OF CLAIMS: 14
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 54 Drawing Figure(s);
241 Drawing Page(s)
LINE COUNT: 4397
CAS INDEXING IS AVAILABLE FOR THIS
PATENT.
AB This invention relates to compositions
comprising a cyclo-oxygenase
modulator in combination with cocoa procyanidin
monomers and/or
oligomers, wherein the cyclo-oxygenase
modulator is a non-steroidal
anti-inflammatory drug such as aspirin. Such
compositions may be used
for the treatment of cardiovascular related
disorders.

CAS INDEXING IS AVAILABLE FOR THIS
PATENT.

L5 ANSWER 9 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2004:4504
USPATFULL
TITLE: Tumor necrosis factor receptor 2
INVENTOR(S): Stanton, Jr., Vincent P.,
Belmont, MA, United States
PATENT ASSIGNEE(S): Nuvelo, Inc., Sunnyvale,
CA, United States (U.S.
corporation)

NUMBER	KIND	DATE

PATENT INFORMATION: US 6673908 B1		
20040106		
APPLICATION INFO.: US 2001-968455		
20011001 (9)		
RELATED APPLN. INFO.: Division of Ser. No. US		
2000-649035, filed on 25 Aug		
2000 Continuation-in-part of Ser. No.		
US 2000-590749,		
filed on 8 Jun 2000, now abandoned		
Continuation-in-part		
of Ser. No. US 2000-495780, filed on 1		
Feb 2000, now		

abandoned Continuation-in-part of Ser.
 No. US 2000-492712, filed on 27 Jan 2000,
 now abandoned
 Continuation-in-part of Ser. No. WO
 2000-US1392, filed
 on 20 Jan 2000 Continuation-in-part of
 Ser. No. US 968455 Continuation-in-part of Ser.
 No. US 1999-451252,
 filed on 29 Nov 1999, now abandoned
 Continuation-in-part of Ser. No. US
 1999-427835, filed
 on 26 Oct 1999, now abandoned
 Continuation-in-part of
 Ser. No. US 1999-414330, filed on 6
 Oct 1999, now
 abandoned Continuation-in-part of Ser.
 No. US 1999-389993, filed on 3 Sep 1999, now
 abandoned
 Continuation-in-part of Ser. No. US
 1999-370841, filed
 on 9 Aug 1999, now abandoned
 Continuation-in-part of
 Ser. No. US 1999-300747, filed on 26
 Apr 1999, now
 abandoned

NUMBER DATE

PRIORITY INFORMATION: US 1999-131334P
 19990426 (60)

US 1999-131191P 19990426 (60)

US 1999-121047P 19990222 (60)

DOCUMENT TYPE: Utility

FILE SEGMENT: GRANTED

PRIMARY EXAMINER: Benzion, Gary

ASSISTANT EXAMINER: Chakrabarti, Arun Kr.

LEGAL REPRESENTATIVE: Fish & Richardson
 P.C.

NUMBER OF CLAIMS: 10

EXEMPLARY CLAIM: 1

NUMBER OF DRAWINGS: 0 Drawing Figure(s);
 0 Drawing Page(s)

LINE COUNT: 17463

CAS INDEXING IS AVAILABLE FOR THIS
 PATENT.

AB The present disclosure describes the use of
 genetic variance information

for genes involved in inflammatory or
 immunologic disease, disorder, or

dysfunction. The variance information is
 indicative of the expected

response of a patient to a method of treatment.

Methods of determining

relevant variance information and additional

methods of using such

variance information are also described.

CAS INDEXING IS AVAILABLE FOR THIS
 PATENT.

L5 ANSWER 10 OF 74 CAPLUS COPYRIGHT
 2005 ACS on STN DUPLICATE 1
 ACCESSION NUMBER: 2004:194249 CAPLUS
 DOCUMENT NUMBER: 140:269493
 TITLE: Inhibition of ***Interleukin*** -
 12 p40
 Transcription and NF-.kappa.B
 Activation by Nitric
 Oxide in Murine Macrophages and
 Dendritic Cells
 AUTHOR(S): Xiong, Huabao; Zhu, Chen; Li,
 Fengling; Hegazi,
 Refaat; He, Kaili; Babyatsky, Mark;
 Bauer, Anthony J.;
 Plevy, Scott E.
 CORPORATE SOURCE: Immunobiology
 Center, The Mount Sinai School of
 Medicine, New York, NY, 10029,
 USA
 SOURCE: Journal of Biological Chemistry
 (2004), 279(11),
 10776-10783
 CODEN: JBCHA3; ISSN: 0021-9258
 PUBLISHER: American Society for
 Biochemistry and Molecular
 Biology
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 AB Nitric oxide (NO), an important effector mol. of
 the innate immune system,
 can also regulate adaptive immunity. In this study,
 the mol. effects of
 NO on the Toll-like receptor signaling pathway
 were detd. using
 interleukin - ***12*** (***IL*** -
 12) as an
 immunol. relevant target gene. The principal
 conclusion of these expts.
 is that ***NO*** ***inhibits*** IL-1
 receptor-assocd. kinase
 (IRAK) activity and attenuates the mol. interaction
 between tumor necrosis
 factor receptor-assocd. factor-6 and IRAK. As a
 consequence, the NO donor
 S-nitroso-N-acetylpenicillamine (SNAP) inhibits
 lipopolysaccharide
 (LPS)-induced ***IL*** - ***12*** p40
 mRNA expression, protein
 prodn., and promoter activity in murine
 macrophages, dendritic cells, and
 the murine macrophage cell line RAW 264.7.
 Splenocytes from inducible
 nitric-oxide synthase-deficient mice demonstrate
 markedly increased
 IL - ***12*** p40 protein and mRNA
 expression compared with wild
 type splenocytes. The inhibitory action of NO on
 IL - ***12***
 p40 is independent of the cytokine IL-10. The
 effects of NO can be

directly attributed to inhibition of NF-.kappa.B activation through IRAK-dependent pathways. Accordingly, SNAP strongly reduces LPS-induced NF-.kappa.B DNA binding to the p40 promoter and inhibits LPS-induced I.kappa.B phosphorylation. Similarly, NO attenuates IL-1.beta.-induced NF-.kappa.B activation. These expts. provide another example of how an innate immune mol. may have a profound effect on adaptive immunity.

REFERENCE COUNT: 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 11 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2003:299854
USPATFULL

TITLE: Combined compositions for tumor vasculature coagulation and treatment

INVENTOR(S): Thorpe, Philip E., Dallas, TX,
UNITED STATES

King, Steven W., Rancho Santa
Margarita, CA, UNITED

STATES
Gottstein, Claudia, Dallas, TX,
UNITED STATES

NUMBER KIND DATE

PATENT INFORMATION: US 2003211075 A1
20031113
APPLICATION INFO.: US 2002-259244 A1
20020927 (10)

NUMBER DATE

PRIORITY INFORMATION: US 2001-325532P
20010927 (60)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Shelley P.M. Fussey,
Ph.D., Williams, Morgan & Amerson,
P.C., Suite 1100, 10333 Richmond
Avenue, Houston, TX,
77042

NUMBER OF CLAIMS: 43
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 4 Drawing Page(s)
LINE COUNT: 9999
CAS INDEXING IS AVAILABLE FOR THIS
PATENT.

AB Disclosed are various defined combinations of agents for use in improved anti-vascular therapies and coagulative tumor treatment. Particularly provided are combined treatment methods, and associated compositions,

pharmaceuticals, medicaments, kits and uses, which together function surprisingly effectively in the treatment of vascularized tumors. The invention preferably involves a component or treatment step that enhances the effectiveness of therapy using targeted or non-targeted coagulants to cause tumor vasculature thrombosis.

CAS INDEXING IS AVAILABLE FOR THIS
PATENT.

L5 ANSWER 12 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2003:288693
USPATFULL
TITLE: IL-17 homologous polypeptides and therapeutic uses thereof

INVENTOR(S): Chen, Jian, Princeton, NJ,
UNITED STATES

Filvaroff, Ellen, San Francisco, CA,
UNITED STATES

Fong, Sherman, Alameda, CA,
UNITED STATES

Goddard, Audrey, San Francisco, CA,
UNITED STATES

Godowski, Paul, Burlingame, CA,
UNITED STATES

Grimaldi, J. Christopher, San
Francisco, CA, UNITED
STATES

Gurney, Austin, Belmont, CA,
UNITED STATES

Li, Hanzhong, San Mateo, CA,
UNITED STATES

Hillan, Kenneth, San Francisco, CA,
UNITED STATES

Hymowitz, Sarah G., San Francisco,
CA, UNITED STATES

Tumas, Daniel, Orinda, CA, UNITED
STATES

Starovasnik, Melissa A., San Francisco,
CA, UNITED
STATES

VanLookeren, Menno, San Francisco,
CA, UNITED STATES

Vandlen, Richard, Hillsborough, CA,
UNITED STATES

Watanabe, Colin, Moraga, CA,
UNITED STATES

Williams, P. Mickey, Half Moon Bay,
CA, UNITED STATES

Wood, William I., Hillsborough, CA,
UNITED STATES

Yansura, Daniel, Pacifica, CA,
UNITED STATES

PATENT ASSIGNEE(S): Genentech, Inc., South
San Francisco, CA, UNITED
STATES, 94080 (U.S. corporation)

NUMBER KIND DATE

PATENT INFORMATION: US 2003203451 A1
20031030

APPLICATION INFO.: US 2003-458442 A1
20030610 (10)

RELATED APPLN. INFO.: Division of Ser. No. US
2001-874503, filed on 5 Jun

2001, PENDING Continuation-in-part
of Ser. No. US

2001-816744, filed on 22 Mar 2001,
GRANTED, Pat. No. US

6579520 Continuation-in-part of Ser.
No. WO

2001-US6520, filed on 28 Feb 2001,
PENDING

Continuation-in-part of Ser. No. US
2000-747259, filed
on 20 Dec 2000, GRANTED, Pat. No.
US 6569645

Continuation-in-part of Ser. No. WO
2000-US23328, filed
on 24 Aug 2000, PENDING

DOCUMENT TYPE: Utility

FILE SEGMENT: APPLICATION

LEGAL REPRESENTATIVE: GENENTECH, INC.,
1 DNA WAY, SOUTH SAN FRANCISCO, CA,
94080

NUMBER OF CLAIMS: 60

EXEMPLARY CLAIM: 1

NUMBER OF DRAWINGS: 55 Drawing Page(s)

LINE COUNT: 8852

CAS INDEXING IS AVAILABLE FOR THIS
PATENT.

AB The present invention is directed to novel
polypeptides having sequence
identity with IL-17, IL-17 receptors and to nucleic
acid molecules

encoding those polypeptides. Also provided
herein are vectors and host

cells comprising those nucleic acid sequences,
chimeric polypeptide

molecules comprising the polypeptides of the
present invention fused to

heterologous polypeptide sequences, antibodies
which bind to the

polypeptides of the present invention and to
methods for producing the

polypeptides of the present invention. Further
provided herein are

methods for treating degenerative cartilaginous
disorders and other
inflammatory diseases.

CAS INDEXING IS AVAILABLE FOR THIS
PATENT.

L5 ANSWER 13 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2003:282701
USPATFULL

TITLE: IL-17 homologous polypeptides
and therapeutic uses
thereof

INVENTOR(S): Chen, Jian, Princeton, NJ,
UNITED STATES

Filvaroff, Ellen, San Francisco, CA,
UNITED STATES

Fong, Sherman, Alameda, CA,
UNITED STATES

Goddard, Audrey, San Francisco, CA,
UNITED STATES

Godowski, Paul, Hillsborough, CA,
UNITED STATES

Grimaldi, Christopher, San Francisco,
CA, UNITED STATES

Gurney, Austin, Belmont, CA,
UNITED STATES

Li, Hanzhong, San Mateo, CA,
UNITED STATES

Hillan, Kenneth, San Francisco, CA,
UNITED STATES

Tumas, Daniel, Orinda, CA, UNITED
STATES

VanLookeren, Menno, San Francisco,
CA, UNITED STATES

Vandlen, Richard, Hillsborough, CA,
UNITED STATES

Watanabe, Colin, Moraga, CA,
UNITED STATES

Williams, P. Mickey, Half Moon Bay,
CA, UNITED STATES

Wood, William I., Hillsborough, CA,
UNITED STATES

Yansura, Daniel, Pacifica, CA,
UNITED STATES

PATENT ASSIGNEE(S): Genentech, Inc., South
San Francisco, CA, UNITED STATES
(U.S. corporation)

NUMBER KIND DATE

PATENT INFORMATION: US 2003199044 A1
20031023

APPLICATION INFO.: US 2003-410552 A1
20030408 (10)

RELATED APPLN. INFO.: Division of Ser. No. US
2000-747259, filed on 20 Dec

2000, GRANTED, Pat. No. US
6569645 Continuation-in-part

of Ser. No. WO 2000-US23328, filed
on 24 Aug 2000,

PENDING

DOCUMENT TYPE: Utility

FILE SEGMENT: APPLICATION

LEGAL REPRESENTATIVE: GENENTECH, INC.,
1 DNA WAY, SOUTH SAN FRANCISCO, CA,
94080

NUMBER OF CLAIMS: 60

EXEMPLARY CLAIM: 1

NUMBER OF DRAWINGS: 47 Drawing Page(s)

LINE COUNT: 8602

CAS INDEXING IS AVAILABLE FOR THIS
PATENT.

AB The present invention is directed to novel
polypeptides and to nucleic

acid molecules encoding those polypeptides. Also provided herein are vectors and host cells comprising those nucleic acid sequences, chimeric polypeptide molecules comprising the polypeptides of the present invention fused to heterologous polypeptide sequences, antibodies which bind to the polypeptides of the present invention and to methods for producing the polypeptides of the present invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 14 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2003:277229
USPATFULL

TITLE: Inhibitors of nitric oxide synthase
INVENTOR(S): Singh, Inderjit, Mount Pleasant, SC, UNITED STATES
PATENT ASSIGNEE(S): MUSC Foundation for Research Development (U.S. corporation)

NUMBER KIND DATE

PATENT INFORMATION: US 2003195256 A1
20031016
APPLICATION INFO.: US 2002-273557 A1
20021018 (10)
RELATED APPLN. INFO.: Division of Ser. No. US
2000-579791, filed on 25 May
2000, GRANTED, Pat. No. US
6511800 Continuation of Ser.
No. WO 1998-US25360, filed on 25
Nov 1998, PENDING

NUMBER DATE

PRIORITY INFORMATION: US 1997-66839P
19971125 (60)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Michael R.
Krawzsenek, FULBRIGHT & JAWORSKI L.L.P.,
Suite 2400, 600 Congress Avenue,
Austin, TX, 78701
NUMBER OF CLAIMS: 75
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 11 Drawing Page(s)
LINE COUNT: 7728
CAS INDEXING IS AVAILABLE FOR THIS
PATENT.
AB The current invention discloses novel methods
for the inhibition of
inducible nitric oxide synthesis (iNOS) and the
production of NO.
Methods of inhibiting the induction of
proinflammatory cytokines are

also described. Methods of treating various
disease states, such as
X-linked adrenoleukodystrophy, multiple
sclerosis, Alzheimer's and
septic shock using inhibitors of iNOS and
cytokine induction are
disclosed. The inhibitors include the exemplary
compounds lovastatin, a
sodium salt of phenylacetic acid (NaPA), FPT
inhibitor II, N-acetyl
cysteine (NAC), and cAMP.

CAS INDEXING IS AVAILABLE FOR THIS
PATENT.

L5 ANSWER 15 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2003:265291
USPATFULL

TITLE: IL-17 homologous polypeptides
and therapeutic uses
thereof

INVENTOR(S): Chen, Jian, Princeton, NJ,
UNITED STATES
Filvaroff, Ellen, San Francisco, CA,
UNITED STATES
Fong, Sherman, Alameda, CA,
UNITED STATES
Goddard, Audrey, San Francisco, CA,
UNITED STATES
Godowski, Paul J., Hillsborough, CA,
UNITED STATES
Grimaldi, Christopher, San Francisco,
CA, UNITED STATES
Gurney, Austin, Belmont, CA,
UNITED STATES
Li, Hanzhong, San Mateo, CA,
UNITED STATES
Hillan, Kenneth, San Francisco, CA,
UNITED STATES
Tumas, Daniel, Orinda, CA, UNITED
STATES
VanLookeren, Menno, San Francisco,
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Vandlen, Richard, Hillsborough, CA,
UNITED STATES
Watanabe, Colin, Moraga, CA,
UNITED STATES
Williams, P. Mickey, Half Moon Bay,
CA, UNITED STATES
Wood, William I., Hillsborough, CA,
UNITED STATES
Yansura, Daniel, Pacifica, CA,
UNITED STATES
PATENT ASSIGNEE(S): Genentech, Inc., South
San Francisco, CA, UNITED STATES
(U.S. corporation)

NUMBER KIND DATE

PATENT INFORMATION: US 2003186306 A1
20031002

APPLICATION INFO.: US 2003-410374 A1
20030408 (10)

RELATED APPLN. INFO.: Division of Ser. No. US
2000-747259, filed on 20 Dec

2000, GRANTED, Pat. No. US
6569645 Continuation-in-part
of Ser. No. WO 2000-US23328, filed
on 24 Aug 2000,

PENDING

DOCUMENT TYPE: Utility

FILE SEGMENT: APPLICATION

LEGAL REPRESENTATIVE: GENENTECH, INC.,
1 DNA WAY, SOUTH SAN FRANCISCO, CA,
94080

NUMBER OF CLAIMS: 60

EXEMPLARY CLAIM: 1

NUMBER OF DRAWINGS: 47 Drawing Page(s)

LINE COUNT: 8095

CAS INDEXING IS AVAILABLE FOR THIS
PATENT.

AB The present invention is directed to novel
polypeptides and to nucleic
acid molecules encoding those polypeptides. Also
provided herein are
vectors and host cells comprising those nucleic
acid sequences, chimeric
polypeptide molecules comprising the
polypeptides of the present
invention fused to heterologous polypeptide
sequences, antibodies which
bind to the polypeptides of the present invention
and to methods for
producing the polypeptides of the present
invention.

CAS INDEXING IS AVAILABLE FOR THIS
PATENT.

L5 ANSWER 16 OF 74 USPATFULL on STN

ACCESSION NUMBER: 2003:258639

USPATFULL

TITLE: 207 human secreted proteins

INVENTOR(S): Ni, Jian, Germantown, MD,
UNITED STATES

Ebner, Reinhard, Gaithersburg, MD,
UNITED STATES

LaFleur, David W., Washington, DC,
UNITED STATES

Moore, Paul A., Germantown, MD,
UNITED STATES

Olsen, Henrik S., Gaithersburg, MD,
UNITED STATES

Rosen, Craig A., Laytonville, MD,
UNITED STATES

Ruben, Steven M., Olney, MD,
UNITED STATES

Soppet, Daniel R., Centreville, VA,
UNITED STATES

Young, Paul E., Gaithersburg, MD,
UNITED STATES

Shi, Yanggu, Gaithersburg, MD,
UNITED STATES

Florence, Kimberly A., Rockville, MD,
UNITED STATES

Wei, Ying-Fei, Berkeley, CA, UNITED
STATES

Florence, Charles, Rockville, MD,
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Hu, Jing-Shan, Mountain View, CA,
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Li, Yi, Sunnyvale, CA, UNITED

STATES

Kyaw, Hla, Frederick, MD, UNITED

STATES

Fischer, Carrie L., Burke, VA,

UNITED STATES

Ferrie, Ann M., Painted Post, NY,

UNITED STATES

Fan, Ping, Potomac, MD, UNITED

STATES

Feng, Ping, Gaithersburg, MD,

UNITED STATES

Endress, Gregory A., Florence, MA,

UNITED STATES

Dillon, Patrick J., Carlsbad, CA,

UNITED STATES

Carter, Kenneth C., North Potomac,

MD, UNITED STATES

Brewer, Laurie A., St. Paul, MN,

UNITED STATES

Yu, Guo-Liang, Berkeley, CA,

UNITED STATES

Zeng, Zhizhen, Lansdale, PA, UNITED

STATES

Greene, John M., Gaithersburg, MD,

UNITED STATES

NUMBER KIND DATE

PATENT INFORMATION: US 2003181692 A1
20030925

APPLICATION INFO.: US 2001-933767 A1
20010822 (9)

RELATED APPLN. INFO.: Continuation-in-part of
Ser. No. WO 2001-US5614, filed
on 21 Feb 2001, PENDING

Continuation-in-part of Ser.

No. US 1998-205258, filed on 4 Dec
1998, PENDING

NUMBER DATE

PRIORITY INFORMATION: US 2000-184836P
20000224 (60)

US 2000-193170P 20000329 (60)

US 1997-48885P 19970606 (60)

US 1997-49375P 19970606 (60)

US 1997-48881P 19970606 (60)

US 1997-48880P 19970606 (60)

US 1997-48896P 19970606 (60)

US 1997-49020P 19970606 (60)

US 1997-48876P 19970606 (60)

US 1997-48895P 19970606 (60)

US 1997-48884P 19970606 (60)

US 1997-48894P 19970606 (60)
 US 1997-48971P 19970606 (60)
 US 1997-48964P 19970606 (60)
 US 1997-48882P 19970606 (60)
 US 1997-48899P 19970606 (60)
 US 1997-48893P 19970606 (60)
 US 1997-48900P 19970606 (60)
 US 1997-48901P 19970606 (60)
 US 1997-48892P 19970606 (60)
 US 1997-48915P 19970606 (60)
 US 1997-49019P 19970606 (60)
 US 1997-48970P 19970606 (60)
 US 1997-48972P 19970606 (60)
 US 1997-48916P 19970606 (60)
 US 1997-49373P 19970606 (60)
 US 1997-48875P 19970606 (60)
 US 1997-49374P 19970606 (60)
 US 1997-48917P 19970606 (60)
 US 1997-48949P 19970606 (60)
 US 1997-48974P 19970606 (60)
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 US 1997-48897P 19970606 (60)
 US 1997-48898P 19970606 (60)
 US 1997-48962P 19970606 (60)
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 US 1997-57645P 19970905 (60)
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 US 1997-57666P 19970905 (60)
 US 1997-57764P 19970905 (60)
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 US 1997-57661P 19970905 (60)
 US 1997-57662P 19970905 (60)
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 US 1997-57649P 19970905 (60)
 US 1997-57770P 19970905 (60)
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 US 1997-57761P 19970905 (60)
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 US 1997-57629P 19970905 (60)
 US 1997-57628P 19970905 (60)
 US 1997-57777P 19970905 (60)

US 1997-57634P 19970905 (60)
 US 1997-70923P 19971218 (60)
 US 1998-92921P 19980715 (60)
 US 1998-94657P 19980730 (60)
 US 1997-70923P 19971218 (60)
 US 1998-92921P 19980715 (60)
 US 1998-94657P 19980730 (60)

DOCUMENT TYPE: Utility
 FILE SEGMENT: APPLICATION
 LEGAL REPRESENTATIVE: HUMAN GENOME
 SCIENCES INC, 9410 KEY WEST AVENUE,
 ROCKVILLE, MD, 20850

NUMBER OF CLAIMS: 23
 EXEMPLARY CLAIM: 1
 NUMBER OF DRAWINGS: 10 Drawing Page(s)
 LINE COUNT: 32746
 CAS INDEXING IS AVAILABLE FOR THIS
 PATENT.

AB The present invention relates to novel human
 secreted proteins and
 isolated nucleic acids containing the coding
 regions of the genes
 encoding such proteins. Also provided are
 vectors, host cells,
 antibodies, and recombinant methods for
 producing human secreted
 proteins. The invention further relates to
 diagnostic and therapeutic
 methods useful for diagnosing and treating
 diseases, disorders, and/or
 conditions related to these novel human secreted
 proteins.

CAS INDEXING IS AVAILABLE FOR THIS
 PATENT.

L5 ANSWER 17 OF 74 USPATFULL on STN
 ACCESSION NUMBER: 2003:257205

USPATFULL
 TITLE: IL-17 homologous polypeptides
 and therapeutic uses
 thereof

INVENTOR(S): Chen, Jian, Princeton, NJ,
 UNITED STATES
 Filvaroff, Ellen, San Francisco, CA,
 UNITED STATES
 Fong, Sherman, Alameda, CA,
 UNITED STATES
 Goddard, Audrey, San Francisco, CA,
 UNITED STATES
 Godowski, Paul, Hillsborough, CA,
 UNITED STATES
 Grimaldi, Christopher, San Francisco,
 CA, UNITED STATES
 Gurney, Austin, Belmont, CA,
 UNITED STATES
 Li, Hanzhong, San Mateo, CA,
 UNITED STATES
 Hillan, Kenneth, San Francisco, CA,
 UNITED STATES
 Tumas, Daniel, Orinda, CA, UNITED
 STATES

VanLookeren, Menno, San Francisco,
CA, UNITED STATES
Vandlen, Richard, Hillsborough, CA,
UNITED STATES
Watanabe, Colin, Moraga, CA,
UNITED STATES
Williams, P. Mickey, Half Moon Bay,
CA, UNITED STATES
Wood, William I., Hillsborough, CA,
UNITED STATES
Yansura, Daniel, Pacifica, CA,
UNITED STATES
PATENT ASSIGNEE(S): Genentech, Inc., South
San Francisco, CA, UNITED
STATES, 94080 (U.S. corporation)

NUMBER	KIND	DATE
PATENT INFORMATION: US 2003180255 A1		
20030925		
APPLICATION INFO.: US 2003-410927 A1		
20030409 (10)		
RELATED APPLN. INFO.: Division of Ser. No. US		
2001-816744, filed on 22 Mar		
2001, GRANTED, Pat. No. US		
6579520 Continuation-in-part		
of Ser. No. WO 2001-US6520, filed on		
28 Feb 2001,		
PENDING Continuation-in-part of Ser.		
No. US		
2000-747259, filed on 20 Dec 2000,		
GRANTED, Pat. No. US		
6569645 Continuation-in-part of Ser.		
No. WO		
2000-US23328, filed on 24 Aug 2000,		
PENDING		
DOCUMENT TYPE: Utility		
FILE SEGMENT: APPLICATION		
LEGAL REPRESENTATIVE: GENENTECH, INC.,		
1 DNA WAY, SOUTH SAN FRANCISCO, CA,		
94080		
NUMBER OF CLAIMS: 60		
EXEMPLARY CLAIM: 1		
NUMBER OF DRAWINGS: 48 Drawing Page(s)		
LINE COUNT: 8657		
CAS INDEXING IS AVAILABLE FOR THIS		
PATENT.		
AB The present invention is directed to novel		
polypeptides having sequence		
identity with IL-17, IL-17 receptors and to nucleic		
acid molecules		
encoding those polypeptides. Also provided		
herein are vectors and host		
cells comprising those nucleic acid sequences,		
chimeric polypeptide		
molecules comprising the polypeptides of the		
present invention fused to		
heterologous polypeptide sequences, antibodies		
which bind to the		
polypeptides of the present invention and to		
methods for producing the		

polypeptides of the present invention. Further
provided herein are
methods for treating degenerative cartilaginous
disorders and other
inflammatory diseases.

CAS INDEXING IS AVAILABLE FOR THIS
PATENT.

L5 ANSWER 18 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2003:237907
USPATFULL
TITLE: Compositions and methods for the
therapy and diagnosis
of colon cancer
INVENTOR(S): King, Gordon E., Shoreline,
WA, UNITED STATES
Meagher, Madeleine Joy, Seattle, WA,
UNITED STATES
Xu, Jiangchun, Bellevue, WA,
UNITED STATES
Secrist, Heather, Seattle, WA, UNITED
STATES
Jiang, Yuqiu, Kent, WA, UNITED
STATES
PATENT ASSIGNEE(S): Corixa Corporation,
Seattle, WA, UNITED STATES, 98104
(U.S. corporation)

NUMBER	KIND	DATE
PATENT INFORMATION: US 2003166064 A1		
20030904		
APPLICATION INFO.: US 2002-99926 A1		
20020314 (10)		
RELATED APPLN. INFO.: Continuation-in-part of		
Ser. No. US 2001-33528, filed		
on 26 Dec 2001, PENDING		
Continuation-in-part of Ser.		
No. US 2001-920300, filed on 31 Jul		
2001, PENDING		

NUMBER	DATE
PRIORITY INFORMATION: US 2001-302051P	
20010629 (60)	
US 2001-279763P 20010328 (60)	
US 2000-223283P 20000803 (60)	
DOCUMENT TYPE: Utility	
FILE SEGMENT: APPLICATION	
LEGAL REPRESENTATIVE: SEED	
INTELLECTUAL PROPERTY LAW GROUP PLLC,	
701 FIFTH	
AVE, SUITE 6300, SEATTLE, WA,	
98104-7092	
NUMBER OF CLAIMS: 17	
EXEMPLARY CLAIM: 1	
LINE COUNT: 8531	
CAS INDEXING IS AVAILABLE FOR THIS	
PATENT.	
AB Compositions and methods for the therapy and	
diagnosis of cancer,	

particularly colon cancer, are disclosed.
Illustrative compositions
comprise one or more colon tumor polypeptides,
immunogenic portions
thereof, polynucleotides that encode such
polypeptides, antigen
presenting cell that expresses such polypeptides,
and T cells that are
specific for cells expressing such polypeptides.
The disclosed
compositions are useful, for example, in the
diagnosis, prevention
and/or treatment of diseases, particularly colon
cancer.

CAS INDEXING IS AVAILABLE FOR THIS
PATENT.

L5 ANSWER 19 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2003:201388
USPATFULL
TITLE: Combined methods for tumor
vasculature coagulation and
treatment
INVENTOR(S): Thorpe, Philip E., Dallas, TX,
UNITED STATES
King, Steven W., Rancho Santa
Margarita, CA, UNITED
STATES
Gottstein, Claudia, Dallas, TX,
UNITED STATES
PATENT ASSIGNEE(S): Board of Regents, The
University of Texas System and
Peregrine Pharmaceuticals, Inc. (U.S.
corporation)

NUMBER	KIND	DATE

PATENT INFORMATION:	US 2003139374	A1
	20030724	
APPLICATION INFO.:	US 2002-259236	A1
	20020927 (10)	

NUMBER	DATE

PRIORITY INFORMATION:	US 2001-325532P
	20010927 (60)
DOCUMENT TYPE:	Utility
FILE SEGMENT:	APPLICATION
LEGAL REPRESENTATIVE:	Shelley P.M. Fussey, Williams, Morgan & Amerson, P.C., Suite 250, 7676 Hillmont, Houston, TX, 77040
NUMBER OF CLAIMS:	43
EXEMPLARY CLAIM:	1
NUMBER OF DRAWINGS:	4 Drawing Page(s)
LINE COUNT:	10003
CAS INDEXING IS AVAILABLE FOR THIS PATENT.	
AB Disclosed are various defined combinations of agents for use in improved	

anti-vascular therapies and coagulative tumor
treatment. Particularly
provided are combined treatment methods, and
associated compositions,
pharmaceuticals, medicaments, kits and uses,
which together function
surprisingly effectively in the treatment of
vascularized tumors. The
invention preferably involves a component or
treatment step that
enhances the effectiveness of therapy using
targeted or non-targeted
coagulants to cause tumor vasculature thrombosis.

CAS INDEXING IS AVAILABLE FOR THIS
PATENT.

L5 ANSWER 20 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2003:187407
USPATFULL
TITLE: Combined methods for tumor
vasculature coaguligand
treatment
INVENTOR(S): Thorpe, Philip E., Dallas, TX,
UNITED STATES
King, Steven W., Rancho Santa
Margarita, CA, UNITED
STATES
Gottstein, Claudia, Dallas, TX,
UNITED STATES
PATENT ASSIGNEE(S): Board of Regents, The
University of Texas System and
Peregrine Pharmaceuticals, Inc. (U.S.
corporation)

NUMBER	KIND	DATE

PATENT INFORMATION:	US 2003129193	A1
	20030710	
APPLICATION INFO.:	US 2002-259227	A1
	20020927 (10)	

NUMBER	DATE

PRIORITY INFORMATION:	US 2001-325532P
	20010927 (60)
DOCUMENT TYPE:	Utility
FILE SEGMENT:	APPLICATION
LEGAL REPRESENTATIVE:	Shelley P.M. Fussey, Williams, Morgan & Amerson, P.C., Suite 250, 7676 Hillmont, Houston, TX, 77040
NUMBER OF CLAIMS:	45
EXEMPLARY CLAIM:	1
NUMBER OF DRAWINGS:	4 Drawing Page(s)
LINE COUNT:	10012
CAS INDEXING IS AVAILABLE FOR THIS PATENT.	
AB Disclosed are various defined combinations of agents for use in improved anti-vascular therapies and coagulative tumor treatment. Particularly	

provided are combined treatment methods, and associated compositions, pharmaceuticals, medicaments, kits and uses, which together function surprisingly effectively in the treatment of vascularized tumors. The invention preferably involves a component or treatment step that enhances the effectiveness of therapy using targeted or non-targeted coagulants to cause tumor vasculature thrombosis.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 21 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2003:180305
USPATFULL

TITLE: Combined compositions for tumor vasculature coagulant and treatment

INVENTOR(S): Thorpe, Philip E., Dallas, TX, UNITED STATES

King, Steven W., Rancho Santa Margarita, CA, UNITED STATES

Gottstein, Claudia, Dallas, TX, UNITED STATES

PATENT ASSIGNEE(S): Board of Regents, The University of Texas System (U.S. corporation)

NUMBER KIND DATE

PATENT INFORMATION: US 2003124132 A1
20030703

APPLICATION INFO.: US 2002-259223 A1
20020927 (10)

NUMBER DATE

PRIORITY INFORMATION: US 2001-325532P
20010927 (60)

DOCUMENT TYPE: Utility

FILE SEGMENT: APPLICATION

LEGAL REPRESENTATIVE: Shelley P.M. Fussey, Ph.D., WILLIAMS, MORGAN & AMERSON,

P.C., Suite 1100, 10333 Richmond Avenue, Houston, TX, 77042

NUMBER OF CLAIMS: 45

EXEMPLARY CLAIM: 1

NUMBER OF DRAWINGS: 4 Drawing Page(s)

LINE COUNT: 10025

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Disclosed are various defined combinations of agents for use in improved anti-vascular therapies and coagulative tumor treatment. Particularly provided are combined treatment methods, and associated compositions,

pharmaceuticals, medicaments, kits and uses, which together function surprisingly effectively in the treatment of vascularized tumors. The invention preferably involves a component or treatment step that enhances the effectiveness of therapy using targeted or non-targeted coagulants to cause tumor vasculature thrombosis.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 22 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2003:165436

USPATFULL

TITLE: Cocoa extract compounds and methods for making and using the same

INVENTOR(S): Romanczyk, Leo J., JR., Hackettstown, NJ, UNITED STATES

Schmitz, Harold H., Branchburg, NJ, UNITED STATES

PATENT ASSIGNEE(S): MARS Incorporated (U.S. corporation)

NUMBER KIND DATE

PATENT INFORMATION: US 2003113290 A1
20030619

APPLICATION INFO.: US 2002-127817 A1
20020422 (10)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2001-776649, filed on 5 Feb

2001, PENDING Continuation of Ser. No. US 1997-831245,

filed on 2 Apr 1997, GRANTED, Pat. No. US 6297273

Continuation-in-part of Ser. No. US 1996-631661, filed

on 2 Apr 1996, ABANDONED

Continuation of Ser. No. US 2000-717893, filed on 21 Nov 2000,

PENDING Continuation of Ser. No. US 1997-831245, filed on 2

Apr 1997, GRANTED, Pat. No. US 6297273

Continuation-in-part of Ser. No. US 1996-631661, filed on 2

Apr 1996, ABANDONED

DOCUMENT TYPE: Utility

FILE SEGMENT: APPLICATION

LEGAL REPRESENTATIVE: CLIFFORD CHANCE US LLP, 200 PARK AVENUE, NEW YORK, NY, 10166

NUMBER OF CLAIMS: 208

EXEMPLARY CLAIM: 1

NUMBER OF DRAWINGS: 258 Drawing Page(s)

LINE COUNT: 6136

CAS INDEXING IS AVAILABLE FOR THIS
PATENT.

AB Disclosed and claimed are cocoa extracts,
compounds, combinations
thereof and compositions containing the same,
such as polyphenols or
procyanidins, methods for preparing such extracts,
compounds and
compositions, as well as uses for them, especially
a polymeric compound
of the formula A.sub.n, wherein A is a monomer
of the formula:
##STR1##

wherein n is an integer from 2 to 18, such that
there is at least one
terminal monomeric unit A, and one or a plurality
of additional
monomeric units;

R is 3-(.alpha.)--OH, 3-(.beta.)--OH, 3-(.alpha.)--
O-sugar, or
3-(.beta.)--O-sugar;

bonding between adjacent monomers takes place
at positions 4, 6 or 8;

a bond of an additional monomeric unit in
position 4 has alpha or beta
stereochemistry;

X, Y and Z are selected from the group consisting
of monomeric unit A,
hydrogen, and a sugar, with the provisos that as to
the at least one
terminal monomeric unit, bonding of the
additional monomeric unit
thereto (the bonding of the additional monomeric
unit adjacent to the
terminal monomeric unit) is at position 4 and
optionally Y=Z=hydrogen;

the sugar is optionally substituted with a phenolic
moiety, at any
position on the sugar, for instance via an ester
bond, and

pharmaceutically acceptable salts or derivatives
thereof (including
oxidation products).

CAS INDEXING IS AVAILABLE FOR THIS
PATENT.

L5 ANSWER 23 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2003:106233
USPATFULL

TITLE: Compositions and methods for the
therapy and diagnosis
of pancreatic cancer

INVENTOR(S): Benson, Darin R., Seattle,
WA, UNITED STATES

Kalos, Michael D., Seattle, WA,
UNITED STATES
Lodes, Michael J., Seattle, WA,
UNITED STATES
Persing, David H., Redmond, WA,
UNITED STATES
Hepler, William T., Seattle, WA,
UNITED STATES
Jiang, Yuqiu, Kent, WA, UNITED
STATES
PATENT ASSIGNEE(S): Corixa Corporation,
Seattle, WA, UNITED STATES, 98104
(U.S. corporation)

NUMBER KIND DATE

PATENT INFORMATION: US 2003073144 A1
20030417
APPLICATION INFO.: US 2002-60036 A1
20020130 (10)

NUMBER DATE

PRIORITY INFORMATION: US 2001-333626P
20011127 (60)

US 2001-305484P	20010712 (60)
US 2001-265305P	20010130 (60)
US 2001-267568P	20010209 (60)
US 2001-313999P	20010820 (60)
US 2001-291631P	20010516 (60)
US 2001-287112P	20010428 (60)
US 2001-278651P	20010321 (60)
US 2001-265682P	20010131 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: SEED
INTELLECTUAL PROPERTY LAW GROUP PLLC,
701 FIFTH

AVE, SUITE 6300, SEATTLE, WA,

98104-7092

NUMBER OF CLAIMS: 17

EXEMPLARY CLAIM: 1

LINE COUNT: 14253

CAS INDEXING IS AVAILABLE FOR THIS
PATENT.

AB Compositions and methods for the therapy and
diagnosis of cancer,

particularly pancreatic cancer, are disclosed.

Illustrative compositions

comprise one or more pancreatic tumor

polypeptides, immunogenic portions

thereof, polynucleotides that encode such

polypeptides, antigen

presenting cell that expresses such polypeptides,

and T cells that are

specific for cells expressing such polypeptides.

The disclosed

compositions are useful, for example, in the

diagnosis, prevention

and/or treatment of diseases, particularly

pancreatic cancer.

CAS INDEXING IS AVAILABLE FOR THIS
PATENT.

L5 ANSWER 24 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2003:78522
USPATFULL

TITLE: IL-17 homologous polypeptides
and therapeutic uses

thereof

INVENTOR(S): Chen, Jian, Princeton, NJ,
UNITED STATES

Filvaroff, Ellen, San Francisco, CA,
UNITED STATES

Fong, Sherman, Alameda, CA,
UNITED STATES

Goddard, Audrey, San Francisco, CA,
UNITED STATES

Godowski, Paul J., Hillsborough, CA,
UNITED STATES

Grimaldi, J. Christopher, San
Francisco, CA, UNITED

STATES

Gurney, Austin, Belmont, CA,
UNITED STATES

Li, Hanzhong, San Mateo, CA,
UNITED STATES

Hillan, Kenneth, San Francisco, CA,
UNITED STATES

Tumas, Daniel, Orinda, CA, UNITED
STATES

VanLookeren, Menno, San Francisco,
CA, UNITED STATES

Vandlen, Richard, Hillsborough, CA,
UNITED STATES

Watanabe, Colin K., Moraga, CA,
UNITED STATES

Williams, P. Mickey, Half Moon Bay,
CA, UNITED STATES

Wood, William I., Hillsborough, CA,
UNITED STATES

Yansura, Daniel, Pacifica, CA,
UNITED STATES

PATENT ASSIGNEE(S): GENENTECH, INC.
(U.S. corporation)

NUMBER KIND DATE

PATENT INFORMATION: US 2003054442 A1
20030320

APPLICATION INFO.: US 2001-908827 A1
20010718 (9)

RELATED APPLN. INFO.: Continuation of Ser. No.
US 1999-311832, filed on 14

May 1999, PENDING Continuation of
Ser. No. US

1999-380138, filed on 25 Aug 1999,
PENDING Continuation

of Ser. No. US 1999-380142, filed on
25 Aug 1999,

ABANDONED Continuation of Ser.
No. US 2000-644848,

filed on 22 Aug 2000, PENDING
Continuation of Ser. No.

US 2000-747259, filed on 20 Dec
2000, PENDING

Continuation of Ser. No. US 2001-
816744, filed on 22

Mar 2001, PENDING Continuation of
Ser. No. US

2001-854208, filed on 10 May 2001,
PENDING Continuation

of Ser. No. US 2001-854280, filed on
10 May 2001,

PENDING

NUMBER DATE

PRIORITY INFORMATION: WO 1999-US5028
19990308

WO 1999-US10733	19990514
WO 1999-US31274	19991230
WO 2000-US4341	20000218
WO 2000-US5601	20000301
WO 2000-US5841	20000302
WO 2000-US7532	20000321
WO 2000-US15264	20000602
WO 2000-US23328	20000824
WO 2000-US30873	20001110
WO 2000-US32678	20001201
WO 2000-US34956	20001220
WO 2001-US6520	20010228
US 1998-85579P	19980515 (60)
US 1998-113621P	19981223 (60)
US 1999-130232P	19990421 (60)
US 1999-131022P	19990426 (60)
US 1999-134287P	19990514 (60)
US 1999-138387P	19990609 (60)
US 1999-172096P	19991223 (60)
US 2000-175481P	20000111 (60)
US 2000-191007P	20000321 (60)
US 2000-213807P	20000622 (60)
US 2000-242837P	20001024 (60)
US 2000-244072P	20001026 (60)

DOCUMENT TYPE: Utility

FILE SEGMENT: APPLICATION

LEGAL REPRESENTATIVE: GENENTECH, INC.,
1 DNA WAY, SOUTH SAN FRANCISCO, CA,
94080

NUMBER OF CLAIMS: 60

EXEMPLARY CLAIM: 1

NUMBER OF DRAWINGS: 47 Drawing Page(s)

LINE COUNT: 8091

CAS INDEXING IS AVAILABLE FOR THIS
PATENT.

AB The present invention is directed to novel
polypeptides and to nucleic

acid molecules encoding those polypeptides. Also
provided herein are

vectors and host cells comprising those nucleic
acid sequences, chimeric

polypeptide molecules comprising the
polypeptides of the present

invention fused to heterologous polypeptide sequences, antibodies which bind to the polypeptides of the present invention and to methods for producing the polypeptides of the present invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 25 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2003:11111
USPATFULL

TITLE: IL-17 homologous polypeptides and therapeutic uses thereof

INVENTOR(S): Chen, Jian, Princeton, NJ,
UNITED STATES

Filvaroff, Ellen, San Francisco, CA,
UNITED STATES

Fong, Sherman, Alameda, CA,
UNITED STATES

Goddard, Audrey, San Francisco, CA,
UNITED STATES

Godowski, Paul J., Burlingame, CA,
UNITED STATES

Grimaldi, Christopher, San Francisco,
CA, UNITED STATES

Gurney, Austin L., Belmont, CA,
UNITED STATES

Li, Hanzhong, San Mateo, CA,
UNITED STATES

Hillan, Kenneth, San Francisco, CA,
UNITED STATES

Tumas, Daniel, Orinda, CA, UNITED
STATES

VanLookeren, Menno, San Francisco,
CA, UNITED STATES

Vandlen, Richard, Hillsborough, CA,
UNITED STATES

Watanabe, Colin, Moraga, CA,
UNITED STATES

Williams, P. Mickey, Half Moon Bay,
CA, UNITED STATES

Wood, William I., Hillsborough, CA,
UNITED STATES

Yansura, Daniel G., Pacifica, CA,
UNITED STATES

PATENT ASSIGNEE(S): GENENTECH, INC.
(U.S. corporation)

NUMBER KIND DATE

PATENT INFORMATION: US 2003008815 A1
20030109

US 6569645 B2 20030527

APPLICATION INFO.: US 2000-747259 A1
20001220 (9)

RELATED APPLN. INFO.: Continuation-in-part of
Ser. No. US 1999-311832, filed

on 14 May 1999, PENDING

Continuation-in-part of Ser.

No. US 2000-644848, filed on 22 Aug
2000, PENDING

Continuation-in-part of Ser. No. WO
2000-US4341, filed

on 18 Feb 2000, UNKNOWN

Continuation-in-part of Ser.

No. WO 2000-US23328, filed on 24
Aug 2000, UNKNOWN

Continuation-in-part of Ser. No. WO
2000-US32678, filed

on 1 Dec 2000, UNKNOWN

Continuation-in-part of Ser. No.

WO 1999-US31274, filed on 30 Dec
1999, UNKNOWN

Continuation-in-part of Ser. No. WO
2000-US7532, filed

on 21 Mar 2000, UNKNOWN

Continuation-in-part of Ser.

No. WO 2000-US5841, filed on 2 Mar
2000, UNKNOWN

Continuation-in-part of Ser. No. WO
2000-US15264, filed

on 2 Jun 2000, UNKNOWN

Continuation-in-part of Ser. No.

WO 2000-US30873, filed on 10 Nov
2000, UNKNOWN

NUMBER DATE

PRIORITY INFORMATION: US 2000-253646P
20001128 (60)

US 1999-172096P 19991223 (60)

US 2000-175481P 20000111 (60)

US 2000-191007P 20000321 (60)

US 2000-213087P 20000620 (60)

US 2000-242837P 20001024 (60)

DOCUMENT TYPE: Utility

FILE SEGMENT: APPLICATION

LEGAL REPRESENTATIVE: GENENTECH, INC.,
1 DNA WAY, SOUTH SAN FRANCISCO, CA,
94080

NUMBER OF CLAIMS: 60

EXEMPLARY CLAIM: 1

NUMBER OF DRAWINGS: 47 Drawing Page(s)

LINE COUNT: 8685

CAS INDEXING IS AVAILABLE FOR THIS
PATENT.

AB The present invention is directed to novel
polypeptides and to nucleic
acid molecules encoding those polypeptides. Also
provided herein are

vectors and host cells comprising those nucleic
acid sequences, chimeric

polypeptide molecules comprising the

polypeptides of the present

invention fused to heterologous polypeptide

sequences, antibodies which

bind to the polypeptides of the present invention

and to methods for

producing the polypeptides of the present

invention.

CAS INDEXING IS AVAILABLE FOR THIS
PATENT.

L5 ANSWER 26 OF 74 USPATFULL on STN

ACCESSION NUMBER: 2003:3511

USPATFULL

TITLE: IL-17 homologous polypeptides
and therapeutic uses
thereof

INVENTOR(S): Chen, Jian, Princeton, NJ,
UNITED STATES

Filvaroff, Ellen, San Francisco, CA,
UNITED STATES

Fong, Sherman, Alameda, CA,
UNITED STATES

Goddard, Audrey, San Francisco, CA,
UNITED STATES

Godowski, Paul, Burlingame, CA,
UNITED STATES

Grimaldi, Christopher, San Francisco,
CA, UNITED STATES

Gurney, Austin, Belmont, CA,
UNITED STATES

Li, Hanzhong, San Mateo, CA,
UNITED STATES

Hillan, Kenneth, San Francisco, CA,
UNITED STATES

Tumas, Daniel, Orinda, CA, UNITED
STATES

VanLookeren, Menno, San Francisco,
CA, UNITED STATES

Vandlen, Richard, Hillsborough, CA,
UNITED STATES

Watanabe, Colin, Moraga, CA,
UNITED STATES

Williams, P. Mickey, Half Moon Bay,
CA, UNITED STATES

Wood, William I., Hillsborough, CA,
UNITED STATES

Yansura, Daniel, Pacifica, CA,
UNITED STATES

PATENT ASSIGNEE(S): GENENTECH, INC.
(U.S. corporation)

NUMBER KIND DATE

PATENT INFORMATION: US 2003003546 A1
20030102

US 6579520 B2 20030617

APPLICATION INFO.: US 2001-816744 A1
20010322 (9)

RELATED APPLN. INFO.: Continuation-in-part of
Ser. No. US 1999-311832, filed
on 14 May 1999, PENDING

NUMBER DATE

PRIORITY INFORMATION: WO 2001-US6520
20010228

WO 2000-US34956 20001220

WO 2000-US32678 20001201

WO 2000-US30873 20001110

WO 2000-US23328 20000824

WO 2000-US15264 20000602

WO 2000-US7532 20000321

WO 2000-US5841 20000302

WO 2000-US5601 20000301

WO 2000-US4341 20000218

WO 1999-US31274 19991230

WO 1999-US10733 19990514

WO 1999-US5028 19990308

US 2000-253646P 20001128 (60)

US 2000-244072P 20001026 (60)

US 2000-242837P 20001024 (60)

US 2000-213807P 20000622 (60)

US 2000-191007P 20000321 (60)

US 2000-175481P 20000111 (60)

US 1999-172096P 19991223 (60)

US 1999-138387P 19990609 (60)

US 1999-134287P 19990514 (60)

US 1999-131022P 19990426 (60)

US 1999-130232P 19990421 (60)

US 1998-113621P 19981223 (60)

US 1998-85579P 19980515 (60)

DOCUMENT TYPE: Utility

FILE SEGMENT: APPLICATION

LEGAL REPRESENTATIVE: GENENTECH, INC.,
1 DNA WAY, SOUTH SAN FRANCISCO, CA,
94080

NUMBER OF CLAIMS: 60

EXEMPLARY CLAIM: 1

NUMBER OF DRAWINGS: 48 Drawing Page(s)

LINE COUNT: 7774

CAS INDEXING IS AVAILABLE FOR THIS
PATENT.

AB The present invention is directed to novel
polypeptides having sequence
identity with IL-17, IL-17 receptors and to nucleic
acid molecules
encoding those polypeptides. Also provided
herein are vectors and host
cells comprising those nucleic acid sequences,
chimeric polypeptide
molecules comprising the polypeptides of the
present invention fused to
heterologous polypeptide sequences, antibodies
which bind to the
polypeptides of the present invention and to
methods for producing the
polypeptides of the present invention. Further
provided herein are
methods for treating degenerative cartilaginous
disorders and other
inflammatory diseases.

CAS INDEXING IS AVAILABLE FOR THIS
PATENT.

L5 ANSWER 27 OF 74 USPATFULL on STN

ACCESSION NUMBER: 2003:337302

USPATFULL

TITLE: Cocoa extract compounds and
methods for making and
using the same

INVENTOR(S): Romanczyk, Jr., Leo J.,
Hackettstown, NJ, United States
Hammerstone, Jr., John F., Nazareth,
PA, United States
Buck, Margaret M., Morristown, NJ,
United States
Post, Laurie S., Great Meadows, NJ,
United States
Cipolla, Giovanni G., Alpha, NJ,
United States
McClelland, Craig A., East
Stroudsburg, PA, United
States
Mundt, Jeff A., Hackettstown, NJ,
United States
Schmitz, Harold H., Branchburg, NJ,
United States
PATENT ASSIGNEE(S): Mars Incorporated,
McLean, VA, United States (U.S.
corporation)

NUMBER KIND DATE

PATENT INFORMATION: US 6670390 B1
20031230
APPLICATION INFO.: US 2000-717893
20001121 (9)
RELATED APPLN. INFO.: Continuation of Ser. No.
US 1997-831245, filed on 2 Apr
1997, now patented, Pat. No. US
6297273
Continuation-in-part of Ser. No. US
1996-631661, filed
on 2 Apr 1996, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Solola, T. A.
LEGAL REPRESENTATIVE: Kelley, Margaret B.,
Clifford Chance Rogers & Wells
NUMBER OF CLAIMS: 26
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 248 Drawing
Figure(s); 232 Drawing Page(s)
LINE COUNT: 4609
CAS INDEXING IS AVAILABLE FOR THIS
PATENT.
AB Disclosed and claimed are cocoa extracts,
compounds, combinations
thereof and compositions containing the same,
such as polyphenols or
procyanidins, methods for preparing such extracts,
compounds and
compositions, as well as uses for them, especially
a polymeric compound
of the formula A.sub.n, wherein A is a monomer
of the formula: ##STR1##

wherein

n is an integer from 2 to 18, such that there is at
least one terminal

monomeric unit A, and one or a plurality of
additional monomeric units;

R is 3-(.alpha.)-OH, 3-(.beta.)-OH, 3-(.alpha.)-O-
sugar, or
3-(.beta.)-O-sugar.

CAS INDEXING IS AVAILABLE FOR THIS
PATENT.

L5 ANSWER 28 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2003:26240
USPATFULL
TITLE: Methods of treating nitric oxide and
cytokine mediated
disorders
INVENTOR(S): Singh, Inderjit, Mount
Pleasant, SC, United States
PATENT ASSIGNEE(S): Medical University of
South Carolina, Charleston, SC,
United States (U.S. corporation)
MUSC Foundation for Research
Development, Charleston,
SC, United States (U.S. corporation)

NUMBER KIND DATE

PATENT INFORMATION: US 6511800 B1
20030128
APPLICATION INFO.: US 2000-579791
20000525 (9)
RELATED APPLN. INFO.: Continuation of Ser. No.
WO 1998-US25360, filed on 25
Nov 1998

NUMBER DATE

PRIORITY INFORMATION: US 1997-66839P
19971125 (60)
DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Gitomer, Ralph
LEGAL REPRESENTATIVE: Fulbright & Jaworski
LLP
NUMBER OF CLAIMS: 50
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 16 Drawing Figure(s);
11 Drawing Page(s)
LINE COUNT: 7562
CAS INDEXING IS AVAILABLE FOR THIS
PATENT.
AB The current invention discloses novel methods
for the inhibition of
inducible nitric oxide synthesis (iNOS) and the
production of NO.
Methods of inhibiting the induction of
proinflammatory cytokines are
also described. Methods of treating various
disease states, such as
X-linked adrenoleukodystrophy, multiple
sclerosis, Alzheimer's and

septic shock using inhibitors of iNOS and cytokine induction are disclosed. The inhibitors include the exemplary compounds lovastatin, a sodium salt of phenylacetic acid (NaPA), FPT inhibitor II, N-acetyl cysteine (NAC), and cAMP. Methods of treating a nitric oxide or cytokine mediated disorder in a cell comprising administering a biologically effective amount of at least one induction suppressor of an inducible nitric oxide synthase or a cytokine is also described.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 29 OF 74 CAPLUS COPYRIGHT
2005 ACS on STN DUPLICATE 2
ACCESSION NUMBER: 2003:908561 CAPLUS
DOCUMENT NUMBER: 140:192445
TITLE: Ifosfamide impairs the
allostimulatory capacity of
human dendritic cells by intracellular
glutathione

depletion
AUTHOR(S): Kuppner, Maria C.; Schamer,
Anabel; Milani, Valeria;
von Hesler, Christoph; Tschoep,
Katharina E.; Heinz,
Oksana; Issels, Rolf D.

CORPORATE SOURCE: Klinikum Grosshadern,
Medical Clinic III,
Ludwig-Maximilians-University,
Munich, Germany

SOURCE: Blood (2003), 102(10), 3668-
3674

CODEN: BLOOAW; ISSN: 0006-

4971

PUBLISHER: American Society of
Hematology

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Ifosfamide, a clin. potent chemotherapeutic agent, causes the depletion of intracellular glutathione (GSH) levels in various cell types. GSH is the major intracellular reductant against oxidative stress.

4-Hydroxyifosfamide (4-OH-IF), the activated form of ifosfamide, depletes GSH levels in T cells and natural killer (NK) cells; this is accompanied

by a decrease in T-cell and NK-cell function. Here we demonstrate for the first time that human monocyte-derived dendritic cells (DCs) express

higher constitutive levels of GSH and are less sensitive to

4-OH-IF-induced GSH depletion than T cells and NK cells. Treatment of DCs

with 4-OH-IF significantly reduced their ability to stimulate allogeneic

T-cell proliferation and interferon-.gamma. (IFN-.gamma.) prodn.

Ifosfamide also decreased DC interleukin-12p70 (IL-12p70) prodn. after stimulation with lipopolysaccharide (LPS) and IFN-.gamma.. The decrease in allostimulatory capacity and in IFN-.gamma. and ***IL*** - ***12***

prodn. correlated with a decrease in intracellular GSH in the DCs. The responses could be restored by reconstituting DC GSH levels with glutathione monoethyl ester (GSH-OEt). 4-OH-IF had ***no***

inhibitory effect on the ability of DCs to present exogenously

added tyrosinase peptide to tyrosinase-specific cytotoxic T lymphocytes

(CTLs). These studies suggest that in cancer patients treated with

ifosfamide, protection strategies based on glutathione reconstitution may enhance DC function.

REFERENCE COUNT: 43 THERE ARE 43
CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS
AVAILABLE IN THE RE FORMAT

L5 ANSWER 30 OF 74 CAPLUS COPYRIGHT
2005 ACS on STN DUPLICATE 3
ACCESSION NUMBER: 2003:970254 CAPLUS
DOCUMENT NUMBER: 140:123097
TITLE: Unique regulation profile of
prostaglandin E1 on

adhesion molecule expression and
cytokine production
in human peripheral blood
mononuclear cells

AUTHOR(S): Takahashi, Hideo Kohka;
Iwagaki, Hiromi; Tamura,
Ryuji; Xue, Dong; Sano, Masahiro;
Mori, Shuji;

Yoshino, Tadashi; Tanaka, Noriaki;
Nishibori, Masahiro

CORPORATE SOURCE: Department of
Pharmacology, Okayama University
Graduate School of Medicine and
Dentistry, Okayama,
Japan

SOURCE: Journal of Pharmacology and
Experimental Therapeutics

(2003), 307(3), 1188-1195

CODEN: JPETAB; ISSN: 0022-3565

PUBLISHER: American Society for
Pharmacology and Experimental
Therapeutics

DOCUMENT TYPE: Journal

LANGUAGE: English

AB In the present study, the authors examd. the effects of prostaglandin E1

(PGE1) on the expression of intercellular adhesion mol. (ICAM)-1, B7.1, B7.2, CD40, and CD40 ligand (CD40L) on peripheral blood mononuclear cells (PBMC) using fluorescence-activated cell sorting anal. as well as its

effects on cytokine prodn. using ELISA. Whereas
no

inhibitor of spontaneous expression of adhesion mols. was

reported, the authors found that PGE1 inhibited spontaneous ICAM-1, B7.2,

and CD40 expression on monocytes in a concn.-dependent manner but had no

effect on the expression of B7.1 and CD40L.

Although interleukin (IL)-18

induced the expression of ICAM-1, B7.2, CD40, and CD40L, PGE1 prevented

IL-18-induced expression of ICAM-1, B7.2, and CD40. The authors examd.

the involvement of five subtypes of PGE1 receptors (IP, EP1, EP2, EP3, and

EP4) in the effect of PGE1 on the expression of these adhesion mols. using

subtype-specific agonists. Among EP receptor agonists, EP2 and EP4

receptor agonists inhibited IL-18-elicited ICAM-1, B7.2, and CD40

expression. ONO-1301 (IP receptor agonist) prevented the expression of

ICAM-1, B7.2, and CD40 regardless of the presence of IL-18 with the same

potency as PGE1. The effect of a combination of ONO-1301 and 11-deoxy

(D)-PGE1 (EP2/EP4 receptor agonist) on ICAM-1, B7.2, and CD40 expression

mimicked that of PGE1. Moreover, PGE1 inhibited the prodn. of ***IL***

- ***12*** and interferon-.gamma. in PBMC in the presence and absence

of IL-18, whereas PGE1 induced IL-10 prodn. In conclusion, IP receptor

and EP2/EP4 receptor play an important role in the action of PGE1 on the

expression of adhesion mols. on monocytes and cytokine prodn.

REFERENCE COUNT: 40 THERE ARE 40

CITED REFERENCES AVAILABLE FOR THIS

RECORD. ALL CITATIONS

AVAILABLE IN THE RE FORMAT

L5 ANSWER 31 OF 74 EMBASE COPYRIGHT

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on STN

ACCESSION NUMBER: 2003056465 EMBASE

TITLE: Cytotoxicity in glioma cells due to

interleukin -

12 and interleukin-18-stimulated macrophages

mediated by interferon-.gamma.-regulated nitric oxide.

AUTHOR: Kito T.; Kuroda E.; Yokota A.; Yamashita U.

CORPORATE SOURCE: Dr. U. Yamashita, Department of Immunology, Univ. of Occup./Environmental Health, School of Medicine, 1-1

Iseigaoka, Yahatanishi-ku, Kitakyusyu 807-8555, Japan.

yama-uki@med.uoeh-u.ac.jp

SOURCE: Journal of Neurosurgery, (1 Feb 2003) 98/2 (385-392).

Refs: 45

ISSN: 0022-3085 CODEN: JONSAC

COUNTRY: United States

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 008 Neurology and Neurosurgery

026 Immunology, Serology and Transplantation

LANGUAGE: English

SUMMARY LANGUAGE: English

AB Object. Interleukin (***IL***)- ***12*** and IL-18 synergistically

mediate antitumor responses through the production of interferon-.gamma.

(IFN.gamma.) by T and natural killer (NK) cells. Recently, it has been

reported that macrophages stimulated with these cytokines also produce

IFN.gamma., which led the authors to investigate the antglioma activity

of macrophages stimulated by the combination of these cytokines in vitro.

Methods. Dish-adherent peritoneal exudate cells, which had been elicited

in thioglycollate broth as a source of macrophages, were used in the

experiment. The murine glioma cell lines VM-glioma and 203G were labeled

with [(3)H]thymidine for a cytotoxicity assay of macrophages. In response

to the combined stimulation by ***IL*** - ***12*** and IL-18,

macrophages expressed potent cytotoxic activity against glioma cells in

association with increasing production of IFN.gamma. and nitric oxide (

NO). ***Inhibitors*** of NO abrogated the cytotoxic activity

of the macrophages, which had been induced by IL-12 and IL-18, despite the

increase in IFN.gamma. production. Neutralization of IFN.gamma. or use of

macrophages obtained from IFN.gamma. gene-knockout mice markedly reduced

not only cytotoxic activity, but also NO production. Depletion of T and NK

cells from the macrophage population, which was achieved using antibody

plus complement treatment, slightly reduced macrophage activities,

suggesting that these are the main effector cells, although T and NK cells may partially participate in this cytotoxicity. Conclusions. Macrophages stimulated with ***IL*** - ***12*** and IL-18 produced IFN. gamma. and NO, which in turn mediated the antiglioma response. Therefore, macrophages as well as T and NK cells play an important role in antitumor responses stimulated by ***IL*** - ***12*** and IL-18.

L5 ANSWER 32 OF 74 CAPLUS COPYRIGHT
2005 ACS on STN DUPLICATE 4
ACCESSION NUMBER: 2003:514704 CAPLUS
DOCUMENT NUMBER: 139:270480
TITLE: Antitumor effects of yeast (NBG, Immunol) and their

clinical significance
AUTHOR(S): Yagita, Akikuni; Maruyama, Shoji; Sukegawa, Yasushi; Takenoshita, Seiichi; Kanazawa, Masashi; Katoh, Ryoji; Hagi, Hiroo; Fujimoto, Shigeyoshi
CORPORATE SOURCE: Institute of Immunotherapy for Cancer, Kinki University, Japan
SOURCE: Biotherapy (Tokyo, Japan) (2003), 17(3), 257-266

CODEN: BITPE9; ISSN: 0914-2223
PUBLISHER: Gan to Kagaku Ryohosha
DOCUMENT TYPE: Journal
LANGUAGE: Japanese
AB The antineoplastic effect of .beta.-1,3/1,6 glucan (basidiomycete prepn.)

is well known. However, the antineoplastic effect of yeast-derived .beta.-1,3/1,6 glucan, such as NBG (Immunol), has been little

investigated. In the present study, we used models of 3LL s.c. tumor grafts in Th1 strain B10 mice (high tumor resistance) and colon 26 s.c.

tumor grafts in Th2 strain BALB/c mice (low tumor resistance) to

investigate the endogenous ***interleukin*** - ***12*** (***IL***

- ***12***) induction ability and antineoplastic effect of NBG. The

results showed antitumor activity in the NBG-administered groups of both

B10 and BALB/c mice, but in terms of endogenous ***IL*** - ***12***

a significantly high productivity was seen in the highly tumor resistant

B10 mice only. No significant difference was seen in the BALB/c mice,

which have a low tumor resistance. NBG (Immunol) was administered for 3

mo or longer to more than 260 patients with advanced or terminal stage

cancer, in whom effects from immunotherapy are not conventionally seen.

The immunol. changes and clin. efficacy were investigated. With addnl.

administration of NBG (Immunol, 6T or more/day), there was a marked

increase in the no. of NK cells, followed by an increase in NKT cells.

While significant differences in ***IL*** - ***12*** productivity

were not seen in all patients, a significant difference was seen when a

comparison was made in effective cases (96 cases) only. The productivity

of interleukin-10 (IL-10), one of the major immunosuppressant cytokines,

was significantly inhibited after the administration of NBG. However,

no ***inhibitory*** action on the angiogenesis promotion

factor VEGF was obsd.

L5 ANSWER 33 OF 74 CAPLUS COPYRIGHT
2005 ACS on STN DUPLICATE 5

ACCESSION NUMBER: 2002:309776 CAPLUS
DOCUMENT NUMBER: 136:319388

TITLE: Methods and compositions for enhancing the

immunostimulatory effect of ***interleukin*** -

12

INVENTOR(S): Trinchieri, Giorgio; Lee, William M. F.; Koblish,

Holly

PATENT ASSIGNEE(S): The Wistar Institute of Anatomy and Biology, USA; The

Trustees of the University of

Pennsylvania

SOURCE: U.S., 19 pp.

CODEN: USXXAM

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	
APPLICATION NO.	DATE		
US 6375944	B1	20020423	US 1999-395038
19990913			
US 2002081277	A1	20020627	US 2002-79068
20020220			
PRIORITY APPLN. INFO.:			US 1998-101698P
P 19980925			
		US 1999-395038	A3
19990913			

AB The invention discloses a method for enhancing the therapeutic and

adjuvant use of ***IL*** - ***12*** by reducing unwanted transient

immunosuppression caused by ***IL*** -
 12 or by high doses
 thereof by co-administering ***IL*** -
 12 with an effective
 amt. of an agent that inhibits or neutralizes nitric
 oxide (NO) in vivo.
 This enhanced vaccine therapy involves co-
 administering the ***IL*** -
 12 adjuvant, a selected vaccine antigen
 and the ***NO***
 inhibiting /neutralizing agent. Addnl., the
 toxicity of ***IL***
 - ***12*** treatment may be reduced by co-
 administering ***IL*** -
 12 with an effective amt. of the
 NO ***inhibiting***
 or neutralizing agent. A therapeutic compn.
 characterized by reduced
 toxicity in mammals contains ***IL*** -
 12, preferably a low
 dose thereof, and an ***NO***
 inhibiting or neutralizing
 agent in a pharmaceutically acceptable carrier. A
 vaccine compn. contains
 an effective adjuvant amt. of ***IL*** -
 12, an effective amt.
 of an ***NO*** ***inhibiting*** or
 neutralizing agent, and an
 effective protective amt. of a vaccine antigen in a
 pharmaceutically
 acceptable carrier.
 REFERENCE COUNT: 34 THERE ARE 34
 CITED REFERENCES AVAILABLE FOR THIS
 RECORD. ALL CITATIONS
 AVAILABLE IN THE RE FORMAT
 L5 ANSWER 34 OF 74 USPATFULL on STN
 ACCESSION NUMBER: 2002:322509
 USPATFULL
 TITLE: IL-17 homologous polypeptides and
 therapeutic uses
 thereof
 INVENTOR(S): Chen, Jian, Princeton, NJ,
 UNITED STATES
 Filvaroff, Ellen, San Francisco, CA,
 UNITED STATES
 Fong, Sherman, Alameda, CA,
 UNITED STATES
 French, Dorothy, Redwood City, CA,
 UNITED STATES
 Goddard, Audrey, San Francisco, CA,
 UNITED STATES
 Godowski, Paul J., Hillsborough, CA,
 UNITED STATES
 Grimaldi, J. Christopher, San
 Francisco, CA, UNITED
 STATES
 Gurney, Austin L., Belmont, CA,
 UNITED STATES
 Hillan, Kenneth J., San Francisco, CA,
 UNITED STATES

Hymowitz, Sarah G., San Francisco,
 CA, UNITED STATES
 Li, Hanzhong, San Mateo, CA,
 UNITED STATES
 Pan, James, Zitobicoke, CANADA
 Starovasnik, Melissa A., San Francisco,
 CA, UNITED
 STATES
 Tumas, Daniel, Orinda, CA, UNITED
 STATES
 Van Lookeren, Menno, San Francisco,
 CA, UNITED STATES
 Vandlen, Richard, Hillsborough, CA,
 UNITED STATES
 Watanabe, Colin K., Moraga, CA,
 UNITED STATES
 Williams, P. Mickey, Half Moon Bay,
 CA, UNITED STATES
 Wood, William I., Hillsborough, CA,
 UNITED STATES
 Yansura, Daniel G., Pacifica, CA,
 UNITED STATES
 PATENT ASSIGNEE(S): GENENTECH, INC.
 (U.S. corporation)

NUMBER	KIND	DATE
PATENT INFORMATION: US 2002182673 A1		
20021205		
APPLICATION INFO.: US 2001-157 A1		
20011030	(10)	
RELATED APPLN. INFO.: Continuation-in-part of		
Ser. No. US 2001-931836, filed		
on 16 Aug 2001, PENDING		
Continuation-in-part of Ser.		
No. US 2001-929404, filed on 13 Aug		
2001, PENDING		
Continuation-in-part of Ser. No. US		
2001-918585, filed		
on 30 Jul 2001, PENDING		
Continuation-in-part of Ser.		
No. US 2001-908827, filed on 18 Jul		
2001, PENDING		
Continuation-in-part of Ser. No. US		
2001-874503, filed		
on 5 Jun 2001, PENDING		
Continuation-in-part of Ser. No.		
US 2001-854280, filed on 10 May		
2001, PENDING		
Continuation-in-part of Ser. No. US		
2001-854208, filed		
on 10 May 2001, PENDING		
Continuation-in-part of Ser.		
No. US 2001-816744, filed on 22 Mar		
2001, PENDING		
Continuation-in-part of Ser. No. US		
2000-747259, filed		
on 20 Dec 2000, PENDING		
Continuation-in-part of Ser.		
No. US 2000-644848, filed on 22 Aug		
2000, PENDING		

Continuation-in-part of Ser. No. US
1999-380142, filed
on 25 Aug 1999, ABANDONED
Continuation-in-part of Ser.
No. US 1999-380138, filed on 25 Aug
1999, PENDING
Continuation-in-part of Ser. No. US
1999-311832, filed
on 14 May 1999, PENDING

NUMBER DATE

PRIORITY INFORMATION: WO 2001-US21735
20010709

WO 2001-US21066	20010629
WO 2001-US19692	20010620
WO 2001-US17800	20010601
WO 2001-US6520	20010228
WO 2000-US34956	20001220
WO 2000-US32678	20001201
WO 2000-US30873	20001110
WO 2000-US23328	20000824
WO 2000-US15264	20000602
WO 2000-US7532	20000321
WO 2000-US5841	20000302
WO 2000-US5601	20000301
WO 2000-US4341	20000218
WO 1999-US31274	19991230
WO 1999-US10733	19990514
WO 1999-US5028	19990308
US 2000-253646P	20001128 (60)
US 2000-244072P	20001026 (60)
US 2000-242837P	20001024 (60)
US 2000-213807P	20000622 (60)
US 2000-191007P	20000321 (60)
US 2000-175481P	20000111 (60)
US 1999-172096P	19991223 (60)
US 1999-138387P	19990609 (60)
US 1999-134287P	19990514 (60)
US 1999-131022P	19990426 (60)
US 1999-130232P	19990421 (60)
US 1998-113621P	19981223 (60)
US 1998-85579P	19980515 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: GENENTECH, INC.,
1 DNA WAY, SOUTH SAN FRANCISCO, CA,
94080

NUMBER OF CLAIMS: 60
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 70 Drawing Page(s)
LINE COUNT: 8889

CAS INDEXING IS AVAILABLE FOR THIS
PATENT.

AB The present invention is directed to novel
polypeptides having sequence
identity with IL-17, IL-17 receptors and to nucleic
acid molecules
encoding those polypeptides. Also provided
herein are vectors and host
cells comprising those nucleic acid sequences,
chimeric polypeptide

molecules comprising the polypeptides of the
present invention fused to
heterologous polypeptide sequences, antibodies
which bind to the
polypeptides of the present invention and to
methods for producing the
polypeptides of the present invention. Further
provided herein are
methods for treating degenerative cartilaginous
disorders and other
inflammatory diseases.

CAS INDEXING IS AVAILABLE FOR THIS
PATENT.

L5 ANSWER 35 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2002:314711
USPATFULL

TITLE: IL-17 homologous polypeptides
and therapeutic uses

thereof

INVENTOR(S): Chen, Jian, Princeton, NJ,
UNITED STATES

Filvaroff, Ellen, San Francisco, CA,
UNITED STATES

Fong, Sherman, Alameda, CA,
UNITED STATES

Goddard, Audrey, San Francisco, CA,
UNITED STATES

Godowski, Paul J., Burlingame, CA,
UNITED STATES

Grimaldi, J. Christopher, San
Francisco, CA, UNITED

STATES

Gurney, Austin, Belmont, CA,
UNITED STATES

Li, Hanzhong, San Mateo, CA,
UNITED STATES

Hillan, Kenneth, San Francisco, CA,
UNITED STATES

Hymowitz, Sarah G., San Francisco,
CA, UNITED STATES

Tumas, Daniel, Orinda, CA, UNITED
STATES

Starvovasnik, Melissa A., San
Francisco, CA, UNITED

STATES

Lookeren, Menno Van, San Francisco,
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Vandlen, Richard, Hillsborough, CA,
UNITED STATES

Watanabe, Colin, Moraga, CA,
UNITED STATES

Williams, P. Mickey, Half Moon Bay,
CA, UNITED STATES

Wood, William I., Hillsborough, CA,
UNITED STATES

Yansura, Daniel G., Pacifica, CA,
UNITED STATES

PATENT ASSIGNEE(S): GENENTECH, INC.
(U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002177188	A1	20021128
APPLICATION INFO.:	US 2001-874503	A1	20010605 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	WO 2001-US6520	20010228

WO 2000-US34956	20001220
WO 2000-US32678	20001201
WO 2000-US30873	20001110
WO 2000-US23328	20000824
WO 2000-US15264	20000602
WO 2000-US7532	20000321
WO 2000-US5841	20000302
WO 2000-US5601	20000301
WO 2000-US4341	20000218
WO 1999-US31274	19991230
WO 1999-US10733	19990514
WO 1999-US5028	19990308
US 2000-253646P	20001128 (60)
US 2000-244072P	20001026 (60)
US 2000-242837P	20001024 (60)
US 2000-213807P	20000622 (60)
US 2000-191007P	20000321 (60)
US 2000-175481P	20000111 (60)
US 1999-172096P	19991223 (60)
US 1999-138387P	19990609 (60)
US 1999-134287P	19990514 (60)
US 1999-131022P	19990426 (60)
US 1999-130232P	19990421 (60)
US 1998-113621P	19981223 (60)
US 1998-85579P	19980515 (60)

DOCUMENT TYPE: Utility
 FILE SEGMENT: APPLICATION
 LEGAL REPRESENTATIVE: GENENTECH, INC.,
 1 DNA WAY, SOUTH SAN FRANCISCO, CA,
 94080

NUMBER OF CLAIMS: 60
 EXEMPLARY CLAIM: 1
 NUMBER OF DRAWINGS: 54 Drawing Page(s)
 LINE COUNT: 8549

CAS INDEXING IS AVAILABLE FOR THIS
 PATENT.

AB The present invention is directed to novel polypeptides having sequence identity with IL-17, IL-17 receptors and to nucleic acid molecules encoding those polypeptides. Also provided herein are vectors and host cells comprising those nucleic acid sequences, chimeric polypeptide molecules comprising the polypeptides of the present invention fused to heterologous polypeptide sequences, antibodies which bind to the polypeptides of the present invention and to methods for producing the

polypeptides of the present invention. Further provided herein are methods for treating degenerative cartilaginous disorders and other inflammatory diseases.

CAS INDEXING IS AVAILABLE FOR THIS
 PATENT.

L5 ANSWER 36 OF 74 USPATFULL on STN
 ACCESSION NUMBER: 2002:288103
 USPATFULL
 TITLE: Use of combretastatin A4 and its prodrugs as an immune enhancing therapy
 INVENTOR(S): Pero, Ronald W., Sandgate, VT, UNITED STATES
 Lee, Francis Y.F., Yardley, PA, UNITED STATES
 Edvardsen, Klaus, Lund, SWEDEN
 Sjogren, Hans Olov, Lund, SWEDEN

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002160973	A1	20021031
	US 6773702	B2	20040810
APPLICATION INFO.:	US 2001-34746	A1	20011226 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-258283P	20001226 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	COOPER & DUNHAM LLP, 1185 Ave. of the Americas, New York, NY, 10036	
NUMBER OF CLAIMS:	14	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	9 Drawing Page(s)	
LINE COUNT:	1160	
AB	A method of treating immune suppression in a warm-blooded animal bearing a tumor, by administering to the animal an amount of combretastatin A4 and/or a prodrug thereof effective to enhance immune responsiveness without causing vascular destruction. Immunotherapy treatment to inhibit or kill tumor cells includes administering to the animal an immune-response-stimulating agent such as a vaccine of tumor cells genetically modified to produce an immune-response-enhancing cytokine while counteracting tumor-induced immune suppression in the animal by administering combretastatin A4 and/or a prodrug thereof.	

L5 ANSWER 37 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2002:272801
USPATFULL
TITLE: Compositions and methods for the
therapy and diagnosis
of colon cancer
INVENTOR(S): Stolk, John A., Bothell, WA,
UNITED STATES
Xu, Jiangchun, Bellevue, WA,
UNITED STATES
Chenault, Ruth A., Seattle, WA,
UNITED STATES
Meagher, Madeleine Joy, Seattle, WA,
UNITED STATES
PATENT ASSIGNEE(S): Corixa Corporation,
Seattle, WA, UNITED STATES, 98104
(U.S. corporation)

NUMBER KIND DATE

PATENT INFORMATION: US 2002150922 A1
20021017
APPLICATION INFO.: US 2001-998598 A1
20011116 (9)

NUMBER DATE

PRIORITY INFORMATION: US 2001-304037P
20010710 (60)
US 2001-279670P 20010328 (60)
US 2001-267011P 20010206 (60)
US 2000-252222P 20001120 (60)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: SEED
INTELLECTUAL PROPERTY LAW GROUP PLLC,
701 FIFTH
AVE, SUITE 6300, SEATTLE, WA,
98104-7092
NUMBER OF CLAIMS: 17
EXEMPLARY CLAIM: 1
LINE COUNT: 9233
CAS INDEXING IS AVAILABLE FOR THIS
PATENT.
AB Compositions and methods for the therapy and
diagnosis of cancer,
particularly colon cancer, are disclosed.
Illustrative compositions
comprise one or more colon tumor polypeptides,
immunogenic portions
thereof, polynucleotides that encode such
polypeptides, antigen
presenting cell that expresses such polypeptides,
and T cells that are
specific for cells expressing such polypeptides.
The disclosed
compositions are useful, for example, in the
diagnosis, prevention
and/or treatment of diseases, particularly colon
cancer.

CAS INDEXING IS AVAILABLE FOR THIS
PATENT.

L5 ANSWER 38 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2002:243051
USPATFULL
TITLE: Compositions and methods for the
therapy and diagnosis
of ovarian cancer
INVENTOR(S): Algate, Paul A., Issaquah,
WA, UNITED STATES
Jones, Robert, Seattle, WA, UNITED
STATES
Harlocker, Susan L., Seattle, WA,
UNITED STATES
PATENT ASSIGNEE(S): Corixa Corporation,
Seattle, WA, UNITED STATES, 98104
(U.S. corporation)

NUMBER KIND DATE

PATENT INFORMATION: US 2002132237 A1
20020919
APPLICATION INFO.: US 2001-867701 A1
20010529 (9)

NUMBER DATE

PRIORITY INFORMATION: US 2000-207484P
20000526 (60)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: SEED
INTELLECTUAL PROPERTY LAW GROUP PLLC,
701 FIFTH
AVE, SUITE 6300, SEATTLE, WA,
98104-7092
NUMBER OF CLAIMS: 11
EXEMPLARY CLAIM: 1
LINE COUNT: 25718
CAS INDEXING IS AVAILABLE FOR THIS
PATENT.
AB Compositions and methods for the therapy and
diagnosis of cancer,
particularly ovarian cancer, are disclosed.
Illustrative compositions
comprise one or more ovarian tumor polypeptides,
immunogenic portions
thereof, polynucleotides that encode such
polypeptides, antigen
presenting cell that expresses such polypeptides,
and T cells that are
specific for cells expressing such polypeptides.
The disclosed
compositions are useful, for example, in the
diagnosis, prevention
and/or treatment of diseases, particularly ovarian
cancer.
CAS INDEXING IS AVAILABLE FOR THIS
PATENT.

L5 ANSWER 39 OF 74 USPATFULL on STN

ACCESSION NUMBER: 2002:242791

USPATFULL

TITLE: Compositions and methods for the therapy and diagnosis

of colon cancer

INVENTOR(S): King, Gordon E., Shoreline, WA, UNITED STATES

Meagher, Madeleine Joy, Seattle, WA, UNITED STATES

Xu, Jiangchun, Bellevue, WA,

UNITED STATES

Secrist, Heather, Seattle, WA, UNITED

STATES

PATENT ASSIGNEE(S): Corixa Corporation, Seattle, WA, UNITED STATES (U.S. corporation)

NUMBER KIND DATE

PATENT INFORMATION: US 2002131971 A1
20020919

APPLICATION INFO.: US 2001-33528 A1
20011226 (10)

RELATED APPLN. INFO.: Continuation-in-part of
Ser. No. US 2001-920300, filed
on 31 Jul 2001, PENDING

NUMBER DATE

PRIORITY INFORMATION: US 2001-302051P
20010629 (60)

US 2001-279763P 20010328 (60)

US 2000-223283P 20000803 (60)

DOCUMENT TYPE: Utility

FILE SEGMENT: APPLICATION

LEGAL REPRESENTATIVE: SEED

INTELLECTUAL PROPERTY LAW GROUP PLLC,
701 FIFTH

AVE, SUITE 6300, SEATTLE, WA,
98104-7092

NUMBER OF CLAIMS: 17

EXEMPLARY CLAIM: 1

LINE COUNT: 8083

CAS INDEXING IS AVAILABLE FOR THIS
PATENT.

AB Compositions and methods for the therapy and diagnosis of cancer,

particularly colon cancer, are disclosed.

Illustrative compositions

comprise one or more colon tumor polypeptides, immunogenic portions

thereof, polynucleotides that encode such

polypeptides, antigen

presenting cell that expresses such polypeptides, and T cells that are

specific for cells expressing such polypeptides.

The disclosed

compositions are useful, for example, in the diagnosis, prevention

and/or treatment of diseases, particularly colon cancer.

CAS INDEXING IS AVAILABLE FOR THIS
PATENT.

L5 ANSWER 40 OF 74 USPATFULL on STN

ACCESSION NUMBER: 2002:165204

USPATFULL

TITLE: Cocoa extract compounds and methods for making and using the same

INVENTOR(S): Romanczyk, Leo J., JR., Hackettstown, NJ, UNITED STATES

Hammerstone, John F., JR., Nazareth, PA, UNITED STATES

Buck, Margaret M., Morristown, NJ, UNITED STATES

Post, Laurie S., Great Meadows, NJ, UNITED STATES

Cipolla, Giovanni G., Alpha, NJ, UNITED STATES

McClelland, Craig A., East Stroudsburg, PA, UNITED STATES

Mundt, Jeff A., Hackettstown, NJ, UNITED STATES

Schmitz, Harold H., Branchburg, NJ, UNITED STATES

PATENT ASSIGNEE(S): Mars, Incorporated (U.S. corporation)

NUMBER KIND DATE

PATENT INFORMATION: US 2002086833 A1
20020704

US 6638971 B2 20031028

APPLICATION INFO.: US 2001-776649 A1
20010205 (9)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1997-831245, filed on 2 Apr

1997, GRANTED, Pat. No. US 6297273 Continuation-in-part of Ser. No. US 1996-631661, filed on 2 Apr 1996,

ABANDONED

DOCUMENT TYPE: Utility

FILE SEGMENT: APPLICATION

LEGAL REPRESENTATIVE: Clifford Chance Rogers & Wells LLP, 200 Park Avenue,

New York, NY, 10166-0153

NUMBER OF CLAIMS: 208

EXEMPLARY CLAIM: 1

NUMBER OF DRAWINGS: 240 Drawing Page(s)

LINE COUNT: 5797

CAS INDEXING IS AVAILABLE FOR THIS
PATENT.

AB Polyphenol-containing compositions, for example cocoa procyanidin

monomer and/or oligomer-containing

compositions, and their use for

inhibiting bacterial growth are disclosed.

Compositions may be used for

human and veterinary animal administration and may be, for example, in a form of a food, a dietary supplement, or a pharmaceutical.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 41 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2002:181713
USPATFULL

TITLE: Cocoa extract compounds and methods for making and using the same

INVENTOR(S): Romanczyk, Jr., Leo J.,
Hackettstown, NJ, United States
PATENT ASSIGNEE(S): Mars Incorporated,
McLean, VA, United States (U.S.
corporation)

NUMBER KIND DATE

PATENT INFORMATION: US 6423743 B1
20020723

APPLICATION INFO.: US 2000-717833
20001121 (9)

RELATED APPLN. INFO.: Continuation-in-part of
Ser. No. US 1997-831245, filed
on 2 Apr 1997, now patented, Pat. No.
US 6297273

Continuation-in-part of Ser. No. US
1996-631661, filed
on 2 Apr 1996, now abandoned

DOCUMENT TYPE: Utility

FILE SEGMENT: GRANTED

PRIMARY EXAMINER: Solola, T. A.

LEGAL REPRESENTATIVE: Kelley, Margaret B.,
Clifford Chance Rogers & Wells

NUMBER OF CLAIMS: 18

EXEMPLARY CLAIM: 1

NUMBER OF DRAWINGS: 246 Drawing
Figure(s); 234 Drawing Page(s)

LINE COUNT: 4656

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Disclosed and claimed are cocoa extracts, compounds, combinations thereof and compositions containing the same, such as polyphenols or procyanidins, methods for preparing such extracts, compounds and compositions, as well as uses for them, especially a polymeric compound of the formula A.sub.n, wherein A is a monomer of the formula: ##STR1##

wherein

n is an integer from 2 to 18, such that there is at least one terminal monomeric unit A, and one or a plurality of additional monomeric units;

R is 3-(.alpha.)-OH, 3-(.beta.)-OH, 3-(.alpha.)-O-sugar, or 3-(.beta.)-O-sugar.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 42 OF 74 CAPLUS COPYRIGHT
2005 ACS on STN DUPLICATE 6
ACCESSION NUMBER: 2002:239413 CAPLUS
DOCUMENT NUMBER: 136:354082
TITLE: Uptake and processing of
Chlamydia trachomatis by

human dendritic cells
AUTHOR(S): Matyszak, Malgosia K.;
Young, Joyce L.; Gaston, J. S.
Hill

CORPORATE SOURCE: Department of
Medicine, Addenbrooke's Hospital,
University of Cambridge Clinical
School, Cambridge, UK

SOURCE: European Journal of
Immunology (2002), 32(3), 742-751

CODEN: EJIMAF; ISSN: 0014-2980

PUBLISHER: Wiley-VCH Verlag GmbH

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Chlamydia trachomatis (CT) causes several sexually transmitted diseases.

In 2-5% of cases, CT infection leads to the development of reactive arthritis. Dendritic cells (DC) are central in T cell priming and the induction of antigen specific immunity. Here the authors have studied the uptake and processing of CT serovar L2 by human DC, and their ability to present CT antigens to both CD4+ and CD8+ T cells. The authors show that

the entry of CT was mediated by the attachment of CT to heparan sulfates

and could be inhibited by heparin. There was

no

inhibition of uptake by an agent which blocks micropinocytosis.

Infecting DC with CT led to their activation and the prodn. of ***IL***

- ***12*** and TNF-.alpha. but not IL-10.

Following invasion, CT was

confined to distinct vacuoles which were visualized with anti-CT

antibodies using confocal microscopy. Unlike with epithelial cells, these

vacuoles did not develop into characteristic

inclusion bodies. In the

first 48 h, CT+ vacuoles were neg. for Lamp-1 and MHC class II. Despite

no obvious co-localization between CT vacuoles and MHC loading

compartments, infected DC efficiently presented CT antigens to CD4+ T

cells. Infected DC also expanded CT specific CD8+ T cells, allowing the authors to generate a no. of CT-reactive CD8+ T cell clones. There is still controversy about the importance of chlamydia-specific CD8+ T cell responses in patients with arthritis. This is largely due to the difficulties in studying CTL responses at the clonal level. The use of DC as antigen-presenting cells should enable more detailed characterization of these CTL responses.

REFERENCE COUNT: 44 THERE ARE 44
CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS
AVAILABLE IN THE RE FORMAT

L5 ANSWER 43 OF 74 CAPLUS COPYRIGHT
2005 ACS on STN DUPLICATE 7
ACCESSION NUMBER: 2003:78466 CAPLUS
DOCUMENT NUMBER: 139:159652
TITLE: In vitro effects of cAMP-elevating
agents and

glucocorticoid either alone or in
combination on the
production of nitric oxide,
interleukin -
12 and interleukin-10 in
IFN-.gamma.- and
LPS-activated mouse peritoneal
macrophages
AUTHOR(S): Mittal, J.; Dogra, N.; Dass, R.;
Majumdar, S.
CORPORATE SOURCE: Institute of Microbial
Technology, Council of
Scientific and Industrial Research,
Chandigarh, 160

036, India
SOURCE: Folia Microbiologica (Prague,
Czech Republic) (2002),
47(6), 709-716
CODEN: FOMIAZ; ISSN: 0015-5632

PUBLISHER: Institute of Microbiology,
Academy of Sciences of the
Czech Republic

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The effects of cAMP-elevating agents, N6-2'-O-
dibutyryl cAMP (Bu2cAMP),
and glucocorticoid (dexamethasone) on the prodn.
of inflammatory mediators

- nitric oxide and ***interleukin*** - ***12***
(***IL*** -
12) and anti-inflammatory mediator
interleukin-10 (IL-10) were

demonstrated in murine peritoneal macrophages.
Inducible nitric oxide
synthase (iNOS) and iNOS mRNA were detected
by northern blot and western
blot, resp. The cAMP elevating agents Bu2cAMP
and prostaglandin E2 each

alone did not show any effect on NO prodn. but
along with IFN-.gamma. and
lipopolysaccharide (LPS) they slightly enhanced
NO prodn. Dexamethasone
inhibited NO prodn. in IFN-.gamma.- and LPS-
treated cells; cAMP elevating
agents interfered with the NO prodn. inhibited by
dexamethasone.

Inhibition was revealed at the mRNA level as well
as at protein level.

Bu2cAMP or dexamethasone either alone or
synergistically inhibited

IL - ***12*** prodn.; Bu2cAMP
interfered with
dexamethasone-mediated inhibition of IL-10 prodn.
in IFN-.gamma.- and
LPS-treated macrophages. The use of
glucocorticoids along with cAMP

elevating agents was beneficial in lowering the
level of inflammatory
mediator ***IL*** - ***12*** and producing
high levels of the
anti-inflammatory mediator IL-10 active in cell
protection. On the other

hand, interference of Bu2cAMP with
dexamethasone-mediated ***NO***
inhibition may have adverse effect.

Therefore, adverse effects
due to cAMP-mediated interference (inhibition)
with NO synthesis may occur
in many inflammatory diseases during combined
drug therapy by

glucocorticoids and cAMP elevating agents.
REFERENCE COUNT: 28 THERE ARE 28
CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS
AVAILABLE IN THE RE FORMAT

L5 ANSWER 44 OF 74 CAPLUS COPYRIGHT
2005 ACS on STN
ACCESSION NUMBER: 2002:744100 CAPLUS
DOCUMENT NUMBER: 138:121545
TITLE: Effects of tumor supernatant of
A549 lung

adenocarcinoma on human monocyte-
derived dendritic
cells

AUTHOR(S): Chen, Xiaobing; Zhao,
Mingyao; Yang, Hongyan; Huang,
Youtian; Zheng, Zhimin; Ma, Junfen;
Tang, Liming;

Dong, Ziming
CORPORATE SOURCE: Department of
Pathophysiology, Basic Medical College,
Zhengzhou University, Zhengzhou,
450052, Peop. Rep.
China

SOURCE: Zhengzhou Daxue Xuebao,
Yixueban (2002), 37(2),
139-143
CODEN: ZDXYBA; ISSN: 1671-6825

PUBLISHER: Zhengzhou Daxue Xuebao,
Yixueban Bianjibu
DOCUMENT TYPE: Journal
LANGUAGE: Chinese
AB Aim: To investigate the effects of tumor
supernatant (TSN) of A549 lung
adenocarcinoma on human monocyte-derived
dendritic cells (MoDC), and to
study whether functional MoDC pulsed with A549
tumor antigen is effective
in inducing antitumor immune response. Methods:
Human TSN, inactivated
TSN and tumor antigen were prep'd. The 2 kinds of
TSN were added to the
MoDC culture medium throughout the whole
culture course or only at the
late stage (the 7th d) in 7 groups with different
culture conditions; MoDC
were pulsed with tumor antigen on the 4th d or not
and were collected on
the 9th d. MoDC phenotypes were analyzed by
flow cytometry (FCM); the
cell apoptosis was obs'd. by PI staining and FCM.
Function was evaluated
by mixed lymphocyte reaction (MLR), cytotoxic T
lymphocyte (CTL) assay and
IL - ***I2*** in the supernatant was
detected by using ELISA.
Results: When MoDC exposed to TSN in the
whole culture course, phenotypes
showed unobviously, the apoptotic cell ratio was
significantly higher,
MLR, CTL, and ***IL*** - ***I2***
decreased significantly. When
MoDC exposed to TSN at the late culture stage or
exposed to inactivated
TSN throughout the whole culture course,
no ***inhibition***
effects as above were demonstrated. When MoDC
were free from TSN and
pulsed with lung adenocarcinoma cell antigen, the
phenotypes and function
showed obviously. Conclusions: TSN could up-
regulate phenotypic
maturation of MoDC, induce apoptosis and inhibit
the antitumor immunity of
mature MoDC. The normal MoDC pulsed with
tumor antigen could induce
higher antitumor immune response in vitro.

L5 ANSWER 45 OF 74 CAPLUS COPYRIGHT
2005 ACS on STN
ACCESSION NUMBER: 2002:421849 CAPLUS
DOCUMENT NUMBER: 137:80
TITLE: Immunopotentiating activity of
nigerooligosaccharides
AUTHOR(S): Yamamoto, Yoshihiro
CORPORATE SOURCE: Research and
Development Section, Takeda Food
Products, Ltd., Itami, 664-0011, Japan
SOURCE: Fragrance Journal (2002), 30(5),
50-58

CODEN: FUJAD7; ISSN: 0288-9803
PUBLISHER: Fureguransu Janaru Sha
DOCUMENT TYPE: Journal; General Review
LANGUAGE: Japanese
AB A review on the enhancement of ***IL*** -
I2 and IFN-.gamma.
formation by nigerooligosaccharides (NOS;
nigerose, nigerosylglucose,
etc.), augmentation of NK activity by NOS, and
immunopotentiating effects
of NOS in murine disease models and humans.
Mice fed a NOS diet showed
longer survival time after the induction of
endogenous infection, and
NOS ***inhibited*** tumor cell
proliferation in mice. Daily
dietary intake of NOS augmented immune
functions in healthy humans and
improved health-related QOL in the healthy elderly
humans.

L5 ANSWER 46 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2001:168156
USPATFULL
TITLE: Use of cocoa solids having high
cocoa polyphenol
content in tableting compositions and
capsule filling
compositions
INVENTOR(S): Romanczyk, Jr., Leo J.,
Hackettstown, NJ, United States
PATENT ASSIGNEE(S): Mars, Inc., McLean, VA,
United States (U.S.
corporation)

NUMBER	KIND	DATE
PATENT INFORMATION: US 6297273 B1		
20011002		
APPLICATION INFO.: US 1997-831245		
19970402 (8)		
DOCUMENT TYPE: Utility		
FILE SEGMENT: GRANTED		
PRIMARY EXAMINER: Tsang, Cecilia		
ASSISTANT EXAMINER: Solola, Taofiq A.		
LEGAL REPRESENTATIVE: Kelley, Margaret		
B.Clifford Chance Rogers & Wells, LLP		
NUMBER OF CLAIMS: 21		
EXEMPLARY CLAIM: 1		
NUMBER OF DRAWINGS: 237 Drawing		
Figure(s); 221 Drawing Page(s)		
LINE COUNT: 4861		
CAS INDEXING IS AVAILABLE FOR THIS		
PATENT.		
AB Disclosed and claimed are cocoa extracts,		
compounds, combinations		
thereof and compositions containing the same,		
such as polyphenols or		
procyanidins, methods for preparing such extracts,		
compounds and		
compositions, as well as uses for them, especially		
a polymeric compound		

of the formula A.sub.n, wherein A is a monomer of the formula: ##STR1##

wherein n is an integer from 2 to 18, such that there is at least one terminal monomeric unit A, and one or a plurality of additional monomeric units;

R is 3-(.alpha.)-OH, 3-(.beta.)-OH, 3-(.alpha.)-O-sugar, or 3-(.beta.)-O-sugar;

bonding between adjacent monomers takes place at positions 4, 6 or 8;

a bond of an additional monomeric unit in position 4 has alpha or beta stereochemistry;

X, Y and Z are selected from the group consisting of monomeric unit A, hydrogen, and a sugar, with the provisos that as to the at least one terminal monomeric unit, bonding of the additional monomeric unit thereto (the bonding of the additional monomeric unit adjacent to the terminal monomeric unit) is at position 4 and optionally Y=Z=hydrogen;

the sugar is optionally substituted with a phenolic moiety, at any position on the sugar, for instance via an ester bond, and

pharmaceutically acceptable salts or derivatives thereof (including oxidation products).

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 47 OF 74 CAPLUS COPYRIGHT
2005 ACS on STN DUPLICATE 8
ACCESSION NUMBER: 2001:867444 CAPLUS
DOCUMENT NUMBER: 136:131323
TITLE: Host response to infection: the role of CpG DNA in induction of cyclooxygenase 2 and nitric oxide synthase 2 in murine macrophages
AUTHOR(S): Ghosh, Dipak K.; Misukonis, Mary A.; Reich, Charles; Pisetsky, David S.; Weinberg, J. Brice
CORPORATE SOURCE: Department of Medicine, Veterans Affairs and Duke University Medical Centers, Durham, NC, 27705, USA
SOURCE: Infection and Immunity (2001), 69(12), 7703-7710
CODEN: INFIBR; ISSN: 0019-9567

PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Depending on sequence, bacterial and synthetic DNAs can activate the host immune system and influence the host response to infection. The purpose of this study was to det. the abilities of various phosphorothioate oligonucleotides with cytosine-guanosine-contg. motifs (CpG DNA) to activate macrophages to produce nitric oxide (NO) and prostaglandin E2 (PGE2) and to induce expression of NO synthase 2 (NOS2) and cyclooxygenase 2 (COX2). As little as 0.3 .mu.g of CpG DNA/mL increased NO and PGE2 prodn. in a dose- and time-dependent fashion in cells of the mouse macrophage cell line J774. NO and PGE2 prodn. was noted by 4 to 8 h after initiation of cultures with the CpG DNA, with the kinetics of NO prodn. induced by CpG DNA being comparable to that induced by a combination of lipopolysaccharide and gamma interferon. CpG DNA-treated J774 cells showed enhanced expression of NOS2 and COX2 proteins as detd. by immunoblotting, with the relative potencies of the CpG DNAs generally corresponding to those noted for the induction of NO and PGE2 prodn. as well as to those noted for the induction of interleukin-6 (IL-6), ***IL*** - ***I2***, and tumor necrosis factor. Exts. from CpG DNA-treated cells converted L-arginine to L-citrulline, but the ***NOS*** ***inhibitor*** NG-monomethyl-L-arginine (NMMA) inhibited this reaction. The COX2-specific inhibitor NS398 inhibited CpG DNA-induced PGE2 prodn. and inhibited NO prodn. to various degrees. The ***NOS*** ***inhibitors*** NMMA, 1400W, and N-iminoethyl-L-lysine effectively blocked NO prodn. and increased the prodn. of PGE2 in a dose-dependent fashion. Thus, analogs of microbial DNA (i.e., CpG DNA) activate mouse macrophage lineage cells for the expression of NOS2 and COX2, with the prodn. of NO and that of PGE2 occurring in an interdependent manner.
REFERENCE COUNT: 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 48 OF 74 EMBASE COPYRIGHT
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on STN

ACCESSION NUMBER: 2001241683 EMBASE
TITLE: Natural killer cells and nitric oxide.
AUTHOR: Cifone M.G.; Ulisse S.; Santoni A.
CORPORATE SOURCE: M.G. Cifone, Department
of Experimental Medicine,
University of L'Aquila, Via Vetoio, 10
Coppito 2, 67100

L'Aquila, Italy. cifone@univaq.it

SOURCE: International
Immunopharmacology, (2001) 1/8 (1513-1524).

Refs: 103

ISSN: 1567-5769 CODEN: IINMBA

PUBLISHER IDENT.: S 1567-5769(01)00095-9

COUNTRY: Netherlands

DOCUMENT TYPE: Journal; General Review

FILE SEGMENT: 026 Immunology, Serology
and Transplantation

030 Pharmacology

LANGUAGE: English

SUMMARY LANGUAGE: English

AB Natural killer (NK) cells and nitric oxide (NO)
are both important

components of the natural or innate immune
response. NK cells are large

granular lymphocytes capable of destroying cells
infected by virus or

bacteria and susceptible tumor cells without prior
sensitization and

restriction by MHC antigens. They are abundant in
blood, spleen, liver and

lungs and are distinct from both T and B

lymphocytes in their circulation

patterns, profile of surface antigens, receptor

repertoire and the way in

which they discriminate between self and non-self.

Uniquely, NK cells

express receptors that can recognize and

discriminate between normal and

altered MHC class I determinants. NK cell

cytotoxic activity is strongly

induced by cytokines such as IL-2 and ***IL***

- ***12***, and this

activation is associated with synthesis of

NO.

Inhibitors of NO synthesis impair NK
cell-mediated target cell

killing, demonstrating a role for NO in NK cell

function. Furthermore, NO

itself can regulate NK cell activation. In this article,
evidence that NO

is a mediator of NK cell-mediated target cell

killing, and that NO is a

regulator of NK cell activation will be reviewed.

Results of NO synthase

gene deletion studies will be discussed, and rodent
and human NK cells

will be compared. .COPYRG. 2001 Elsevier

Science B.V.

L5 ANSWER 49 OF 74 BIOSIS COPYRIGHT (c)
2005 The Thomson Corporation. on
STN

ACCESSION NUMBER: 2001:258354 BIOSIS

DOCUMENT NUMBER: PREV200100258354

TITLE: Inhibition of ***interleukin*** -

12 (***IL*** - ***12***) p40

transcription by nitric oxide

(NO) in murine macrophages.

AUTHOR(S): Xiong, Huabao [Reprint author];

Zhu, Chen [Reprint author];

Plevy, Scott E. [Reprint author]

CORPORATE SOURCE: Mount Sinai School of

Medicine, One Gustave L. Levy Place,

New York, NY, 10029, USA

SOURCE: FASEB Journal, (March 8, 2001)

Vol. 15, No. 5, pp. A1037.

print.

Meeting Info.: Annual Meeting of the

Federation of American

Societies for Experimental Biology on

Experimental Biology

2001. Orlando, Florida, USA. March 31-

April 04, 2001.

CODEN: FAJOEC. ISSN: 0892-6638.

DOCUMENT TYPE: Conference; (Meeting)

Conference; Abstract; (Meeting Abstract)

LANGUAGE: English

ENTRY DATE: Entered STN: 30 May 2001

Last Updated on STN: 19 Feb 2002

AB Macrophage-derived NO is an important effector
molecule of the innate

immune system. During an immune response, NO
may influence adaptive

immunity. Therefore, we studied whether

macrophage and dendritic cell

derived NO can alter the expression of ***IL***

- ***12***. In a

dose-dependent manner, S-nitroso-N-

acetylpenicillamine (SNAP), an NO

donor, inhibited LPS-induced ***IL*** -

12 p40 and p35 mRNA

expression (RNase protection assay) and

IL - ***12*** p40

protein production (ELISA) in bone marrow

derived murine macrophages,

dendritic cells, and the murine macrophage cell line

RAW 264.7. In a RAW

264.7 derivative that does not produce NO,

increased ***IL*** -

12 p40 mRNA was detected. The

IL - ***12*** p40

promoter is regulated through two important cis-

acting control elements

that bind NF-kB and C/EBP family members.

SNAP inhibited LPS-induced

IL - ***12*** p40 promoter activity in

RAW264.7 cells as

determined by luciferase assay. This strong

inhibitory effect was also

detected using a minimal promoter from -101 to +55 that contains the C/EBP and downstream elements, but not the NF-kB site. In nuclear extracts from LPS-activated RAW 264.7 cells, electrophoretic mobility shift assays revealed that SNAP pretreatment strongly reduced C/EBP and NF-kB DNA binding to the p40 promoter. As IL-10 can potentially inhibit ***IL*** - ***12***, the effects of NO on IL-10 expression was studied. ***NO*** inhibited*** IL-10 mRNA accumulation and promoter activity in RAW 264.7 cells. In IL-10 deficient mice, NO strongly inhibited ***IL*** - ***12*** protein production from macrophages, demonstrating that the inhibitory effects of NO are independent of IL-10. In summary, these results indicate that NO is a potent inhibitor of ***IL*** - ***12*** p40 gene expression in macrophages and dendritic cells. NO may inhibit ***IL*** - ***12*** p40 transcription by attenuating NF-kB and C/EBP activation. These experiments provide another example of how an innate immune effector may have a profound effect on adaptive immunity.

L5 ANSWER 50 OF 74 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
 ACCESSION NUMBER: 2001369454 EMBASE
 TITLE: Nitric oxide and the immune response.
 AUTHOR: Bogdan C.
 CORPORATE SOURCE: C. Bogdan, Institute of Clinical Microbiology, F.-A.-Univ. Erlangen-Nuremberg, Wasserturmstrasse 3-5, D-91054 Erlangen, Germany.
 christian.bogdan@mikro.bio.med.uni-erlangen.de
 SOURCE: Nature Immunology, (2001) 2/10 (907-916).

Refs: 189
 ISSN: 1529-2908 CODEN: NIAMCZ
 COUNTRY: United States
 DOCUMENT TYPE: Journal; General Review
 FILE SEGMENT: 005 General Pathology and Pathological Anatomy
 016 Cancer
 026 Immunology, Serology and Transplantation
 029 Clinical Biochemistry
 037 Drug Literature Index
 LANGUAGE: English
 SUMMARY LANGUAGE: English
 AB During the past two decades, nitric oxide (NO) has been recognized as one

of the most versatile players in the immune system. It is involved in the pathogenesis and control of infectious diseases, tumors, autoimmune processes and chronic degenerative diseases. Because of its variety of reaction partners (DNA, proteins, low-molecular weight thiols, prosthetic groups, reactive oxygen intermediates), its widespread production (by three different NO synthases (NOS) and the fact that its activity is strongly influenced by its concentration, NO continues to surprise and perplex immunologists. Today, there is no simple, uniform picture of the function of NO in the immune system. Protective and toxic effects of NO are frequently seen in parallel. Its striking inter- and intracellular signaling capacity makes it extremely difficult to predict the effect of ***NOS*** ***inhibitors*** and NO donors, which still hampers therapeutic applications.

L5 ANSWER 51 OF 74 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 9
 ACCESSION NUMBER: 2001:598559 CAPLUS
 DOCUMENT NUMBER: 135:287309
 TITLE: Effects of prostaglandin E2 and nitric oxide inhibitors on the expression of interleukin-10, ***interleukin*** - ***12*** and MHC class-II molecules in Mycobacterium microti-infected and interferon-gamma-treated mouse peritoneal macrophages
 AUTHOR(S): Mittal, J.; Dogra, N.; Vohra, H.; Majumdar, S.
 CORPORATE SOURCE: Institute of Microbial Technology, Chandigarh, 160 036, India
 SOURCE: Folia Microbiologica (Prague, Czech Republic) (2001), 46(3), 259-264
 CODEN: FOMIAZ; ISSN: 0015-5632
 PUBLISHER: Institute of Microbiology, Academy of Sciences of the Czech Republic
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 AB Mycobacterium microti-infected mouse peritoneal macrophages produced high ams. of prostaglandin E2 (PGE2) and nitric oxide (NO) when activated with interferon-gamma. (IFN-gamma.). In order to understand the relation

between PGE2 and NO prodn. and the expression of ***interleukin*** -
 12 (***IL*** - ***12***),
 interleukin-10 (IL-10) and MHC
 class-II (Ia) mols. by M. microti-infected and IFN-
 .gamma.-stimulated
 macrophages, we analyzed the level of these mols.
 in the presence or
 absence of PGE2 and ***NO***
 inhibitors . Addn. of
 NG-methyl-L-arginine (L-NMA) and indomethacin
 (IM) caused a significant
 increase in ***IL*** - ***12*** level (2.6-
 and 1.9-fold, resp.)
 whereas IL-10 level decreased by 88 and 56%,
 resp., relative to M.
 microti-infected and IFN-.gamma.-treated control
 macrophages. Enhanced
 PGE2 and NO upregulated IL-10 expression and
 down-regulated ***IL*** -
 12 and MHC class-II (Ia) expression in
 M. microti-infected and
 IFN-.gamma.-treated mouse peritoneal
 macrophages.
 REFERENCE COUNT: 23 THERE ARE 23
 CITED REFERENCES AVAILABLE FOR THIS
 RECORD. ALL CITATIONS
 AVAILABLE IN THE RE FORMAT

L5 ANSWER 52 OF 74 EMBASE COPYRIGHT
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 on STN
 ACCESSION NUMBER: 2002182701 EMBASE
 TITLE: Angiogenesis: From the molecular
 mechanisms to the
 development of new drugs.
 AUTHOR: Morbidelli L.; Donnini S.;
 D'Amore V.; Ziche M.
 CORPORATE SOURCE: L. Morbidelli, Istituto di
 Scienze Farmacologiche,
 Universita di Siena, Siena, Italy
 SOURCE: Acta Medica Romana, (2001) 39/2
 (238-246).

Refs: 24
 ISSN: 0001-6098 CODEN: AMROBA
 COUNTRY: Italy
 DOCUMENT TYPE: Journal; Conference Article
 FILE SEGMENT: 005 General Pathology and
 Pathological Anatomy
 030 Pharmacology
 037 Drug Literature Index
 LANGUAGE: English
 SUMMARY LANGUAGE: English; Italian
 AB The steps required for new vessel growth are
 biologically complex and
 require coordinate regulation of contributing
 components, including
 modifications of cell-cell interactions, proliferation
 and migration of
 endothelial cells and matrix degradation. The
 observation that in vivo

angiogenesis is accompanied by vasodilation, that
 many angiogenesis
 effectors possess vasodilating properties and that
 tumor vasculature is in
 a persistent state of vasodilation, support the
 existence of a
 molecular/biochemical link between vasodilation
 and angiogenesis. Several
 pieces of evidence converge in the indication of a
 role for nitric oxide
 (NO), the factor responsible for vasodilation, in
 physiological and
 pathological angiogenesis. Data originated in
 different labs indicate that
 NO can act both as an "actor" of angiogenesis and
 as a "director of
 angiogenesis", both functions being equally
 expressed during physiological
 and pathological processes. NO significantly
 contributes to the
 prosurvival/proangiogenic program of capillary
 endothelium by triggering
 and transducing cell growth and differentiation via
 endothelial-
 constitutive NO synthase (ec-NOS) activation,
 cyclic GMP (cGMP) elevation,
 mitogen activated kinase (MAPK) activation and
 fibroblast growth factor-2
 (FGF-2) expression. Re-establishment of a
 balanced NO production in the
 cardiovascular system results in a reduction of cell
 damage during
 inflammatory and vascular diseases. Elevation of
 NOS activity in
 correlation with angiogenesis and tumor
 progression has been extensively
 reported in experimental and human tumors. Tumor
 expansion and edema
 formation are sensitive to ***NOS***
 inhibition . On this
 basis, the nitric oxide pathway appears to be a
 promising target for
 consideration in pro- and antiangiogenic
 therapeutic strategies. The use
 of ***NOS*** ***inhibitors*** seems
 appropriate to reduce edema,
 block angiogenesis and facilitate antitumor drug
 delivery.

L5 ANSWER 53 OF 74 BIOSIS COPYRIGHT (c)
 2005 The Thomson Corporation. on
 STN
 ACCESSION NUMBER: 2001:246868 BIOSIS
 DOCUMENT NUMBER: PREV200100246868
 TITLE: Modulation of nitric oxide synthase
 and cyclooxygenase 2 by
 CpG DNA in murine macrophages.
 AUTHOR(S): Ghosh, Dipak K. [Reprint author];
 Misukonis, Mary [Reprint
 author]; Mast, Molly [Reprint author];
 Reigh, Charles

[Reprint author]; Pisetsky, David [Reprint author];

Weinberg, Joe Brice [Reprint author]
CORPORATE SOURCE: Medicine/Hem-oncology,
Duke university and VA Medical

center, 508 fulton street, Durham, NC,
27705, USA

SOURCE: FASEB Journal, (March 7, 2001)
Vol. 15, No. 4, pp. A200.

print.
Meeting Info.: Annual Meeting of the
Federation of American
Societies for Experimental Biology on
Experimental Biology
2001. Orlando, Florida, USA. March 31-
April 04, 2001.

CODEN: FAJOEC. ISSN: 0892-6638.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)

LANGUAGE: English
ENTRY DATE: Entered STN: 23 May 2001

Last Updated on STN: 19 Feb 2002
AB Bacterial and synthetic DNA can act an immune
stimulators and induce

inflammation in vivo. The purpose of this study
was to determine the
abilities of various phosphorothioated, cytosine
guanosine-containing DNAs
("CpG-DNA") to activate mouse macrophages for
nitric oxide (NO) and
prostaglandin E2 (PGE2) production and inducible
NO synthase (NOS2) and
cyclooxygenase (COX2) expression. As little as
0.3 ug/ml CpG-DNA
increased NO and PGE2 production in a dose- and
time-dependent fashion.

An oligonucleotide containing 2 CpG sequences
("SAK2") was generally the
most potent. NO and PGE2 production was noted
by 4 to 8 hours after
initiation of cultures with CpG-DNA, with kinetics
of NO production for
CpG-DNA being comparable to that induced by
LPS/IFN-gamma. LPS,
IFN-gamma, or LPS/IFN-gamma did not induce
PGE2 production. J774 cells
treated with CpG-DNA had enhanced expression of
NOS2 and COX2 protein as
determined by immunoblot, with the relative
potencies of the DNAs
corresponding to that noted for induction of NO
and PGE2 production, as
well as that for induction of IL-6, ***IL*** -
12, and TNF.

Extracts from CpG-DNA-treated cells converted L-
arginine to L-citrulline,
and this was inhibited by the ***NOS***
inhibitor NMMA. The
COX2-specific inhibitor NS398 completely
inhibited CpG-DNA-induced PGE2
production, but had no effect on NO production.
The ***NOS***

inhibitors NMMA, 1400W, and L-NIL
effectively blocked NO
production, but increased production of PGE2 in a
dose-dependent fashion.

Thus, CpG-DNA activates mouse macrophages for
expression of NOS2 and COX2
and production of the pro-inflammatory mediators
NO and PGE2.

L5 ANSWER 54 OF 74 CAPLUS COPYRIGHT
2005 ACS on STN DUPLICATE 10
ACCESSION NUMBER: 2000:466559 CAPLUS
DOCUMENT NUMBER: 133:191684

TITLE: ***Interleukin*** - ***12***
(***IL*** -

12) enhancement of the
cellular immune

response against human
immunodeficiency virus type 1
env antigen in a DNA prime/vaccinia
virus boost

vaccine regimen is time and dose
dependent:

suppressive effects of ***IL*** -
12 boost

are mediated by nitric oxide

AUTHOR(S): Gherardi, M. Magdalena;
Ramirez, Juan C.; Esteban,
Mariano

CORPORATE SOURCE: Department of
Molecular and Cellular Biology, Centro
Nacional de Biotecnologia, CSIC,
Universidad Autonoma,
Madrid, E-28049, Spain

SOURCE: Journal of Virology (2000),
74(14), 6278-6286

CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for

Microbiology
DOCUMENT TYPE: Journal

LANGUAGE: English
AB The authors previously demonstrated that
codelivery of ***interleukin***

- ***12*** (***IL*** - ***12***) with the
human immunodeficiency

virus type 1 (HIV-1) Env antigen from a
recombinant vaccinia virus (rVV)
can enhance the specific anti-Env cell-mediated
immune (CMI) response.

Here, they investigated the effects of ***IL*** -
12 in mice

when it is expressed in a DNA prime/VV boost
vaccine regimen. The

delivery of ***IL*** - ***12*** and Env
product during priming with

a DNA vector, followed by a booster with VV
expressing the Env gene

(rVVenv), was found to trigger the optimal CMI
response compared with

other immunization schedules studied.

Significantly, if ***IL*** -

12 is also delivered as a booster from the viral vector, an impairment of the effects of ***IL*** - ***12*** was obsd. involving nitric oxide (NO), since it was overcome by specific inhibitors of inducible NO synthase. NO caused transient immunosuppression rather than impairment of viral replication. Moreover, at certain viral doses, coadministration of the ***NO*** ***inhibitor*** during the booster resulted in ***IL*** - ***12*** - mediated enhancement of the specific CD8+ T-cell response. In addn., the dose of the ***IL*** - ***12*** -encoding plasmid (pIL-12) and the route of administration of both vectors were relevant factors for optimal CMI responses. Maximal nos. of Env-specific CD8+ .gamma. interferon-secreting cells were obtained when 50 .mu.g of pIL-12 was administered i.m. at priming, followed by an i.v. rVVenv boost. The authors' results demonstrate, in a murine model, crit. parameters affecting the success of vaccination schedules based on a combination of DNA and VV vectors in conjunction with immunomodulators.

REFERENCE COUNT: 59 THERE ARE 59 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 55 OF 74 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 11
 ACCESSION NUMBER: 2000:603722 CAPLUS
 DOCUMENT NUMBER: 133:280428
 TITLE: Babesia bovis-stimulated macrophages express interleukin-1.beta., ***interleukin*** - ***12*** , tumor necrosis factor alpha, and nitric oxide and inhibit parasite replication in vitro

AUTHOR(S): Shoda, Lisl K. M.; Palmer, Guy H.; Florin-Christensen, Jorge; Florin-Christensen, Monica; Godson, Dale L.; Brown, Wendy C.
 CORPORATE SOURCE: Program in Vector-Borne Diseases, Department of Veterinary Microbiology and Pathology, Washington State University, Pullman, WA, 99164-7040, USA
 SOURCE: Infection and Immunity (2000), 68(9), 5139-5145
 CODEN: INFIBR; ISSN: 0019-9567
 PUBLISHER: American Society for Microbiology

DOCUMENT TYPE: Journal
 LANGUAGE: English
 AB The tick-transmitted hemoparasite Babesia bovis causes an acute infection that results in persistence and immunity against challenge infection in cattle that control the initial parasitemia. Resoln. of acute infection with this protozoal pathogen is believed to be dependent on products of activated macrophages (M.PHI.), including inflammatory cytokines and nitric oxide (NO) and its derivs. B. bovis stimulates inducible nitric oxide synthase (iNOS) and prodn. of NO in bovine M.PHI., and chem. donors of. ***NO*** ***inhibit*** the growth of B. bovis in vitro.

However, the induction of inflammatory cytokines in M.PHI. by babesial parasites has not been described, and the antiparasitic activity of NO produced by B. bovis-stimulated M.PHI. has not been definitively demonstrated. We report that monocyte-derived M.PHI. activated by B. bovis expressed enhanced levels of inflammatory cytokines interleukin-1.beta. (IL-1.beta.), ***IL*** - ***12*** , and tumor necrosis factor alpha that are important for stimulating innate and acquired immunity against protozoal pathogens. Furthermore, a lipid fraction of B. bovis-infected erythrocytes stimulated iNOS expression and NO prodn. by M.PHI.. Cocultures of M.PHI. and B. bovis-infected erythrocytes either in contact or phys. sepd. resulted in reduced parasite viability. However, NO produced by bovine M.PHI. in response to B. bovis-infected erythrocytes was only partially responsible for parasite growth inhibition, suggesting that addnl. factors contribute to the inhibition of B. bovis replication. These findings demonstrate that B. bovis induces an innate immune response that is capable of controlling parasite replication and that could potentially result in host survival and parasite persistence.

REFERENCE COUNT: 45 THERE ARE 45 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 56 OF 74 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
 on STN DUPLICATE
 12

ACCESSION NUMBER: 2000249268 EMBASE
TITLE: Targeting activated lymphocytes with
an entirely human

immunotoxin analogue: Human
pancreatic RNase1-human

IL - ***12*** fusion.

AUTHOR: Psarras K.; Ueda M.; Tanabe M.;
Kitajima M.; Aiso S.;

Komatsu S.; Seno M.

CORPORATE SOURCE: Dr. M. Ueda, Department
of Surgery, Keio University School
of Medicine, 35 Shinanomachi, Tokyo
160-8582, Japan

SOURCE: Cytokine, (2000) 12/6 (786-790).

Refs: 15

ISSN: 1043-4666 CODEN: CYTIE

COUNTRY: United Kingdom

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 026 Immunology, Serology
and Transplantation

029 Clinical Biochemistry

LANGUAGE: English

SUMMARY LANGUAGE: English

AB A hybrid human protein was produced in E. coli
by fusing the genes

encoding human pancreatic RNase1 (hpRNase1)
and human IL-2 (hIL-2). The

recombinant hpRNase1-hIL-2 inhibited protein
synthesis in HTLV-1-infected,

malignant T cells, which hyperproduce high
affinity IL-2 receptors, with

an IC50 of 2×10^{-8} M, whereas ***no***

inhibition was

detectable in control cells with lower affinity
receptors. HpRNase1 alone

had an IC50 of almost 10^{-3} M. A molar excess of
hIL-2 blocked the protein

synthesis inhibition dose-dependently. In a human
mixed lymphocyte

culture, hpRNase1-hIL-2 inhibited the proliferation
of responder cells

with potency comparable to that of cyclosporine,
while non-effective doses

of FK506 importantly improved its potency.

Despite its short half-life in

animals, hpRNase1-hIL-2 rapidly enters cells in a
few minutes and arrests

the protein translation in less than 10 h. Thus,
hpRNase1-hIL-2 may be

useful to selectively eliminate activated

lymphocytes hyperproducing high

affinity IL-2 receptors, as in allograft rejection,
graft-versus-host

disease, autoimmune disorders, adult T cell
leukaemia and other

lymphoproliferative or retroviral malignancies
including HIV infection,

without inducing general immunosuppression. As
an entirely human

'immunotoxin analogue' it may alleviate the dose
limiting toxicity and

immunogenicity of conventional immunotoxins.
(C) 2000 Academic Press.

L5 ANSWER 57 OF 74 BIOSIS COPYRIGHT (c)
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STN DUPLICATE 13

ACCESSION NUMBER: 2000:428915 BIOSIS

DOCUMENT NUMBER: PREV200000428915

TITLE: ***Interleukin*** - ***12***

enhances the antitumor

activity of cytotoxic T lymphocytes
against lung

adenocarcinoma engrafted in severe
combined immunodeficient

mice.

AUTHOR(S): Hanagiri, Takeshi [Reprint
author]; Imahayashi, Satoru;

Yoshino, Ichiro; So, Tomoko; Eifuku,

Ryozo; Yoshimatsu,

Takashi; Takenoyama, Mitsuhiro; Osaki,

Toshihiro;

Nakanishi, Ryoich; Ichiyoshi, Yuji;

Nomoto, Kikuo;

Yasumoto, Kosei

CORPORATE SOURCE: Second Department of
Surgery, School of Medicine,

University of Occupational and

Environmental Health, 1-1

Iseigaoka, Yahatanishi-ku, Kitakyushu,
807-8555, Japan

SOURCE: International Journal of Clinical
Oncology, (August, 2000)

Vol. 5, No. 4, pp. 262-268. print.

ISSN: 1341-9625.

DOCUMENT TYPE: Article

LANGUAGE: English

ENTRY DATE: Entered STN: 4 Oct 2000

Last Updated on STN: 10 Jan 2002

AB Background: Through a number of biologic
activities, ***interleukin***

12 (***IL*** - ***12***) has

proven to be a potential

antitumor cytokine in mice bearing a variety of
malignancies. However, in

clinical trials in humans, the eradication of solid
tumors remains

difficult. Methods: A lung cancer cell line (PC-9)-
specific cytotoxic T

lymphocytes (CTL) were generated by multiple
stimulations, with irradiated

PC-9 cells, of regional lymph node lymphocytes
obtained from patients with

lung cancer whose cells expressed the same HLA-
A locus haplotype as PC-9

(HLA-A24). Severe combined immunodeficient
(SCID) mice bearing a

subcutaneous graft of PC-9 were then

intravenously injected with

anti-PC-9-specific CTLs. Under these conditions,
the in-vivo effect of

recombinant human (rh) IL-2 and rh ***IL*** -
12 was

evaluated, based on tumor growth. Results: Mice that received either rh

IL-2 or rh ***IL*** - ***12*** exhibited ***no***

inhibitory effect on tumor growth.

However, mice that received

adoptive immunotherapy (AIT) alone exhibited a significant inhibition of

tumor growth in the PC-9 graft in comparison to untreated mice. When mice

were treated with AIT combined with rh IL-2 + rh ***IL*** - ***12***

administration, tumor growth was significantly suppressed. A significant

difference was observed in the growth of the PC-9 graft between AIT + IL-2

+ ***IL*** - ***12*** treatment and AIT + IL-2 treatment. Four of

eight mice in the AIT + IL-2 + ***IL*** - ***12*** -treated group

showed complete tumor regression. Conclusion: ***IL*** - ***12***

showed a synergistic effect with adoptive immunotherapy, using CTL in a

tumor-engrafted SCID model. These results are therefore considered to

provide a sufficient rationale for IL-2 + ***IL*** - ***12*** -based

immunotherapy using CTL transfer for patients with lung cancer.

L5 ANSWER 58 OF 74 CAPLUS COPYRIGHT

2005 ACS on STN DUPLICATE 14

ACCESSION NUMBER: 2000:868538 CAPLUS

DOCUMENT NUMBER: 135:14036

TITLE: Differential anti-inflammatory and anti-oxidative

effects of dexamethasone and N-acetylcysteine in

endotoxin-induced lung inflammation

AUTHOR(S): Rocksen, D.; Lilliehook, B.;

Larsson, R.; Johansson,

T.; Bucht, A.

CORPORATE SOURCE: Department of

Biomedicine, Defence Research

Establishment, Umea, SE-90182,

Swed.

SOURCE: Clinical and Experimental

Immunology (2000), 122(2),

249-256

CODEN: CEXIAL; ISSN: 0009-9104

PUBLISHER: Blackwell Science Ltd.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Inhalation of bacterial endotoxin induces an acute inflammation in the

lower respiratory tract. In this study, the anti-inflammatory effects of

the anti-oxidant N-acetylcysteine (NAC) and the glucocorticoid

dexamethasone were investigated in mice exposed to aerosolized endotoxin

(lipopolysaccharide (LPS)). Powerful redn. of neutrophils in

bronchoalveolar lavage fluid (BALF) was obtained by a single i.p.

injection of dexamethasone (10 mg/kg), whereas treatment with NAC only

resulted in redn. of neutrophils when administered at a high dose (500

mg/kg). Measurement of cytokine and chemokine expression in lung tissue

revealed a decrease of tumor necrosis factor-alpha, IL-1.alpha.,

IL-1.beta., IL-6, IL-12p40, and MIP-1.alpha.

mRNA when mice were treated

with dexamethasone but not when treated with

NAC. Anal. of oxidative

burst demonstrated a remarkable redn. of oxygen radicals in BALF

neutrophils after treatment with dexamethasone, whereas the effect of NAC

was not different from that in untreated animals. In conclusion,

dexamethasone exerted both anti-inflammatory and anti-oxidative effects in

acute airway inflammation, probably by blocking early events in the

inflammatory cascade. In contrast, treatment with NAC resulted in a weak

redn. of the inflammatory response but ***no*** ***inhibition***

of pro-inflammatory cytokines or redn. of oxidative burst in neutrophils.

These results demonstrate dramatic differences in efficiency and also

indicate that the 2 drugs have different actions.

Combined treatment with

NAC and dexamethasone revealed an additive action but no synergy was obsd.

REFERENCE COUNT: 27 THERE ARE 27

CITED REFERENCES AVAILABLE FOR THIS

RECORD. ALL CITATIONS

AVAILABLE IN THE RE FORMAT

L5 ANSWER 59 OF 74 CAPLUS COPYRIGHT

2005 ACS on STN DUPLICATE 15

ACCESSION NUMBER: 2001:605707 CAPLUS

DOCUMENT NUMBER: 136:198507

TITLE: Phagocytosis of bacteria by mouse bone marrow-derived

dendritic cells affects their ability to process a

heterologous soluble antigen in vitro

AUTHOR(S): Bryniarski, Krzysztof;

Biedron, Rafal; Petrovska,

Liliana; Free, Paul; Chain, Benjamin;

Marcinkiewicz,

Janusz

CORPORATE SOURCE: Department of

Immunology, Jagiellonian University

Medical College, Krakow, 31-121,

Pol.

SOURCE: Central European Journal of Immunology (2000), 25(4), 210-215
CODEN: CJIMFW; ISSN: 1426-3912

PUBLISHER: Termedia
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Sentinel dendritic cells are likely to encounter both live and dead bacteria at sites of infection. Although dendritic cells can phagocytose such bacteria, their principle role is not in bacterial killing but in stimulation of a subsequent adaptive immune response. In contrast, neutrophils at the site of infection play a major role in bacterial killing, in part via oxidative chlorination by myeloperoxidase products. In this study, the interaction between bacterial phagocytosis and the antigen processing function of dendritic cells is examd. Ingestion of heat-killed Salmonella typhimurium, or bacteria killed by oxidative chlorination, induces up-regulation of co-stimulatory mols. on dendritic cells, and strong stimulation of the TH1-inducing proinflammatory cytokines ***IL*** - ***I2*** and TNF-.alpha.. In contrast, induction of nitric oxide prodn. is weak. Finally, phagocytosis of bacteria inhibits processing of protein antigen, but only if phagocytosis precedes exposure to antigen by 24 h. Phagocytosis itself has ***no*** ***inhibitory*** effect on the concomitant processing and presentation of either protein or peptide antigen to T cells. These results demonstrate that both phagocytic and antigen processing pathways can operate simultaneously within dendritic cells, allowing these sentinel cells to operate effectively at the site of bacterial infection.

REFERENCE COUNT: 21 THERE ARE 21 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 60 OF 74 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 16
ACCESSION NUMBER: 2000:102656 CAPLUS
DOCUMENT NUMBER: 132:249760
TITLE: Neutralizing antibodies to granulocyte-macrophage colony-stimulating factor, interleukin-1.alpha. and interferon-.alpha. but not other cytokines in human

immunoglobulin preparations

AUTHOR(S): Wadhwa, M.; Meager, A.; Dilger, P.; Bird, C.; Dolman, C.; Das, R. G.; Thorpe, R.

CORPORATE SOURCE: Division of Immunobiology, National Institute for Biological Standards and Control, Potters Bar, EN6 3QG, UK

SOURCE: Immunology (2000), 99(1), 113-123
CODEN: IMMUAM; ISSN: 0019-2805

PUBLISHER: Blackwell Science Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Human Ig preps. are used therapeutically for various disorders. Such therapy is generally safe but adverse effects occasionally occur in recipients. It has been suggested that antibodies to cytokines present in clin. Ig products may contribute to undesirable effects in recipients. Therefore, we investigated i.v. and i.m. Ig products for the presence of cytokine-specific neutralizing antibodies. Using validated bioassays, we detected neutralizing activity against human granulocyte-macrophage colony-stimulating factor (GM-CSF), interferon-.alpha.2a (IFN-.alpha.2a) and interleukin-1.alpha. (IL-1.alpha.) in Ig products. We found ***no*** ***neutralization*** of granulocyte colony-stimulating factor, macrophage colony-stimulating factor, stem cell factor, IL-1.beta., IL-2, IL-3, IL-4, IL-6, IL-9, IL-10, ***IL*** - ***I2***, tumor necrosis factor-.alpha., oncostatin M (OSM) and IFN-.gamma.. Most batches which neutralized IFN-.alpha.2a activity also neutralized other IFN-.alpha. subtypes, IFN-.omega. and IFN-.beta.. Most products (94%) neutralized the biol. activity of GM-CSF. No correlation between batches and their ability to neutralize bioactivities of GM-CSF, IFN-.alpha.2a and IL-1.alpha. was found. This neutralizing activity could be traced to plasma pools used for manuf. of Igs. The neutralization was mediated by specific cytokine antibodies contained within Ig products as it was present in specific IgG fractions eluted from cytokine affinity chromatog. columns. Specific binding of such IgG fractions to cytokines in immunoblots and in enzyme-linked immunosorbent assays (ELISAs) was obsd.

This contrasts with the broad non-specific recognition of cytokine proteins obsd. using unfractionated Igs in ELISAs. This is the first comprehensive study showing the presence of neutralizing antibodies against GM-CSF, IL-1.alpha., or IFN-.alpha.2a in Ig products.
 REFERENCE COUNT: 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 61 OF 74 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 17
 ACCESSION NUMBER: 1999:510658 CAPLUS
 DOCUMENT NUMBER: 131:125825
 TITLE: Effects of nitric oxide on the induction and differentiation of Th1 cells
 AUTHOR(S): Niedbala, Wanda; Wei, Xiao-Qing; Piedrafito, David; Xu, Damo; Liew, Foo Yew
 CORPORATE SOURCE: Dep. Immunology, Univ. Glasgow, Glasgow, G11 6NT, UK
 SOURCE: European Journal of Immunology (1999), 29(8), 2498-2505
 CODEN: EJIMAF; ISSN: 0014-2980
 PUBLISHER: Wiley-VCH Verlag GmbH
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 AB The authors have previously shown that mice lacking inducible NO synthase are markedly more susceptible to Leishmania major infection but developed a significantly enhanced Th1 cell response compared with wild-type mice. At high concns., ***NO*** ***inhibited*** ***IL*** - ***12*** synthesis by activated macrophages, thereby indirectly suppressing the expansion of Th1 cells. We report that at low concns., NO selectively enhanced the induction of Th1 cells and had no effect on Th2 cells. NO exerted this effect in synergy with ***IL*** - ***12*** during Th1 cell differentiation and had no effect on fully committed Th1 cells. NO appears to affect CD4+ T cells directly and not at the antigen-presenting cells. These results therefore provide an addnl. pathway by which NO modulates the immune response and contributes to the homeostasis of the immune system.
 REFERENCE COUNT: 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 62 OF 74 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 18
 ACCESSION NUMBER: 1999:376749 CAPLUS
 DOCUMENT NUMBER: 131:30804
 TITLE: The mucosal adjuvant effects of cholera toxin and immune-stimulating complexes differ in their requirement for ***IL*** - ***12***, indicating different pathways of action
 AUTHOR(S): Grdic, Dubravka; Smith, Rosemary; Donachie, Anne; Kjerrulf, Martin; Hornquist, Elisabeth; Mowat, Allan; Lycke, Nils
 CORPORATE SOURCE: Department Medical Microbiology Immunology, Univ. Goteborg, Goteborg, S-41346, Swed.
 SOURCE: European Journal of Immunology (1999), 29(6), 1774-1784
 CODEN: EJIMAF; ISSN: 0014-2980
 PUBLISHER: Wiley-VCH Verlag GmbH
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 AB Adjuvants that can improve mucosal vaccine efficacy are much warranted. Studying cholera toxin (CT) and immune-stimulating complexes (ISCOM) the authors found that, contrary to CT, ovalbumin (QVA)-ISCOM were poor inducers of mucosal anti-OVA IgA responses, but induced similar or better systemic immunity following oral immunizations. The addn. of CT to the oral OVA-ISCOM protocol did not stimulate local anti-OVA IgA immunity, nor did it change the quality or magnitude of the systemic responses. Both vectors recruited strong innate immunity, but only OVA-ISCOM could directly induce ***IL*** - ***12***, demonstrable at the protein and mRNA levels. CT had ***no*** ***inhibitory*** effects on lipopolysaccharide/IFN-.gamma.-induced ***IL*** - ***12*** mRNA expression or ***IL*** - ***12*** prodn. Adjuvanticity of CT was unaffected in ***IL*** - ***12*** -deficient mice, while OVA-ISCOM showed partly impaired adjuvant effects by the lack of ***IL*** - ***12***. CT abrogated the induction of oral tolerance stimulated by antigen feeding in these mice. CT did not alter TGF-.beta. levels, suggesting that the immunomodulating effect of CT was independent of ***IL*** - ***12*** as well as TGF-.beta. prodn. These findings

indicate that mucosal adjuvanticity of CT and ISCOM are differently dependent on ***IL*** - ***12***, suggesting that sep. and distinct antigen-processing pathways are involved.
REFERENCE COUNT: 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 63 OF 74 MEDLINE on STN DUPLICATE 19
ACCESSION NUMBER: 2000099817 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10634000
TITLE: Depressed cytolytic activity of peripheral blood

mononuclear cells in unusually high paclitaxel concentrations: reversal by IL-2 and ***IL*** - ***12***

AUTHOR: Chen Y M; Yang W K; Ting C C; Yang D M; Whang-Peng J; Perng R P

CORPORATE SOURCE: Department of Chest Department, Taipei Veterans General Hospital, Taiwan, ROC.

SOURCE: Zhonghua yi xue za zhi = Chinese medical journal; Free China ed, (1999 Dec) 62 (12) 867-74.
Journal code: 0005327. ISSN: 0578-1337.

PUB. COUNTRY: China
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200001

ENTRY DATE: Entered STN: 20000209
Last Updated on STN: 20000209
Entered Medline: 20000128

AB BACKGROUND: Human lymphocyte function was inhibited by high concentrations of paclitaxel and the effect was reversed by interleukin (IL)-2. However, there was no parallel study determining the relationship between paclitaxel concentrations in the lymphocyte cultures and pharmacokinetic analysis in human patients, nor was there any study on the reversal by cytokines, other than IL-2, of the paclitaxel-induced suppression of lymphocyte cytotoxicity. METHODS: We tested the effect of different doses of paclitaxel with various incubation times on the cytolytic activity of peripheral blood mononuclear cells (PBMNCs) against K-562 target cells.

RESULTS: Our results showed that using a schedule similar to that for treating patients with tolerable doses of paclitaxel, ***no***

inhibition of cytolytic activity of PBMNCs was seen. When the paclitaxel concentration was increased 10-fold, the cytolytic activity of PBMNCs was significantly reduced. This suppression was reversed by the simultaneous addition of a low dose (10 U/ml) of IL-2 or ***IL*** - ***12***. Addition of granulocyte macrophage-colony stimulating factor (10 U/ml) did not affect the cytolytic activity of PBMNCs, whereas addition of IL-4 reduced it. Time kinetic studies revealed that, with the addition of IL-2 or ***IL*** - ***12***, most of the mononuclear cellular cytolytic activity recovered within 48 to 72 hours. CONCLUSIONS: These findings suggested that, to reduce the toxicity on mononuclear cellular function when high-dose paclitaxel treatment is elected in clinical practice, paclitaxel should be infused over a longer duration of time, or the treatment should be combined with the administration of a low dose of IL-2 or ***IL*** - ***12***.

L5 ANSWER 64 OF 74 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 20
ACCESSION NUMBER: 1999:409846 CAPLUS
DOCUMENT NUMBER: 131:227568
TITLE: Mechanisms of Cytokine-Mediated Inhibition of Viral

Replication
AUTHOR(S): Komatsu, Takashi; Srivastava, Neil; Revzin, Margarita; Ireland, Derek D. C.; Chesler, David; Shoshkes Reiss, Carol

CORPORATE SOURCE: Department of Biology, New York University, New York

City, NY, 10003-6688, USA
SOURCE: Virology (1999), 259(2), 334-341

CODEN: VIRLAX; ISSN: 0042-6822
PUBLISHER: Academic Press
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Here, the role of nitric oxide synthase (NOS) and ***IL*** - ***12***

administration in inhibition of vesicular stomatitis virus (VSV) from infected neuroblastoma cells was examd. The authors previously have shown that cytokine treatment of cells results in the induction of NOS-1, and this is assocd. with a 2 log inhibition of VSV prodn. The authors performed these studies to examine the mechanism by which viral

replication is suppressed. Neuroblastoma cells (NB41A3) were treated with either ***IL*** - ***12*** or medium and subsequently infected with VSV. Viral protein and mRNA were isolated from these cells, and their levels were measured by Western or Northern blots, resp. mRNA levels were decreased modestly, but viral proteins were decreased substantially in cells pretreated with ***IL*** - ***12***, suggesting that the inhibitory effect of NO is working at the translational level. Cytokine treatment of cells was not assocd. with oxidative stress. The viral proteins also were nitrosylated. Apparently, the mechanism of ***NO***

inhibition of viral replication occurs via translational interference and posttranslational modifications of viral components. (c) 1999 Academic Press.

REFERENCE COUNT: 64 THERE ARE 64
CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS
AVAILABLE IN THE RE FORMAT

L5 ANSWER 65 OF 74 CAPLUS COPYRIGHT
2005 ACS on STN DUPLICATE 21
ACCESSION NUMBER: 1998:800538 CAPLUS
DOCUMENT NUMBER: 130:51318
TITLE: Nitric oxide regulates Th1 cell
development through

the inhibition of ***IL*** -
12 synthesis
by macrophages

AUTHOR(S): Huang, Fang-Ping; Niedbala,
Wanda; Wei, Xiao-Qing; Xu,
Damo; Feng, Gui-Jie; Robinson, John
H.; Lam, Charles;
Liew, Foo Y.

CORPORATE SOURCE: Department
Immunology, University Glasgow, Glasgow,
G11 6NT, UK

SOURCE: European Journal of
Immunology (1998), 28(12),
4062-4070

CODEN: EJIMAF; ISSN: 0014-2980

PUBLISHER: Wiley-VCH Verlag GmbH

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The authors have previously reported that mice
lacking inducible NO

synthase (NOS2) developed enhanced Th1 cell
responses. The authors now

investigated the mechanism by which NO
modulates Th1 cells

differentiation. Peritoneal macrophages from

NOS2-deficient mice infected

with Leishmania major in vivo or stimulated with
interferon(IFN)-.gamma.

or lipopolysaccharide (LPS) in vitro produced
higher levels of
interleukin(***IL***)- ***12*** than those
from heterozygous or
wild-type mice. A macrophage cell line, J774,
produced significant amts.

of ***IL*** - ***12*** following activation
with LPS, or LPS +

IFN-.gamma.. This was markedly enhanced by the
NOS

inhibitor L-NG monomethyl Arg (L-
NMMA), but profoundly inhibited
by the NO-generating compd. S-nitroso-N-acetyl-
penicillamine (SNAP). The

effect of NO in this system is selective, since

SNAP enhanced and L-NMMA

decreased TNF-.alpha. synthesis by LPS-activated
J774 cells. The

differential effect of NO on ***IL*** -
12 and TNF-.alpha. is

at the transcriptional level and is activation
dependent. Since

IL - ***12*** is a major inducer of Th1
cells which produce

IFN-.gamma. that can activate macrophages to
produce ***IL*** -

12, these data demonstrate that NO can
be an inhibitor of this

feedback loop, preventing the excessive
amplification of Th1 cells which

are implicated in a range of immunopathologies.

REFERENCE COUNT: 41 THERE ARE 41
CITED REFERENCES AVAILABLE FOR THIS

RECORD. ALL CITATIONS
AVAILABLE IN THE RE FORMAT

L5 ANSWER 66 OF 74 CAPLUS COPYRIGHT
2005 ACS on STN DUPLICATE 22

ACCESSION NUMBER: 1998:717539 CAPLUS

DOCUMENT NUMBER: 130:80197

TITLE: Immune suppression by
recombinant interleukin (rIL)-12

involves interferon .gamma. induction
of nitric oxide

synthase 2 (iNOS) activity: inhibitors
of NO

generation reveal the extent of rIL-12
vaccine

adjuvant effect

AUTHOR(S): Koblish, Holly Kurzawa;

Hunter, Christopher A.;

Wysocka, Maria; Trinchieri, Giorgio;

Lee, William M.

F.

CORPORATE SOURCE: Cell and Molecular
Biology Graduate Group, Cancer

Center, and Institute for Human Gene
Therapy, School

of Medicine, University of

Pennsylvania, Philadelphia,

PA, 19104, USA

SOURCE: Journal of Experimental
Medicine (1998), 188(9),
1603-1610
CODEN: JEMEA; ISSN: 0022-1007

PUBLISHER: Rockefeller University Press
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Recombinant ***interleukin*** ***12*** (
IL - ***12***)
can profoundly suppress cellular immune responses
in mice. To define the
underlying mechanism, recombinant murine (rm)
IL - ***12*** was
given to C57BL/6 mice undergoing
alloimmunization and found to transiently
but profoundly suppress in vivo and in vitro
allogeneic responses and in
vitro splenocyte mitogenic responses. Use of
neutralizing antibodies and
genetically deficient mice showed that IFN-
gamma. (but not TNF-.alpha.)
mediated rmIL-12-induced immunosuppression.
Splenocyte fractionation
studies revealed that adherent cells from rmIL-12-
treated mice suppressed
the mitogenic response of normal nonadherent cells
to Con A and IL-2.
Addn. of an inhibitor of nitric oxide synthase
(NOS) restored mitogenic
responses, and inducible (i)NOS-/- mice were not
immunosuppressed by
rmIL-12. These results support the view that
suppression of T cell
responses is due to NO produced by macrophages
responding to the high
levels of IFN-gamma: induced by rmIL-12. When
a ***NOS***
inhibitor was given with rmIL-12 during
vaccination of A/J mice
with irradiated SCK tumor cells,
immunosuppression was averted and the
extent of rmIL-12's ability to enhance induction of
protective antitumor
immunity was revealed. This demonstrates that
rmIL-12 is an effective
vaccine adjuvant whose efficacy may be masked by
its transient
immunosuppressive effect.

REFERENCE COUNT: 31 THERE ARE 31
CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS
AVAILABLE IN THE RE FORMAT

L5 ANSWER 67 OF 74 CAPLUS COPYRIGHT
2005 ACS on STN DUPLICATE 23
ACCESSION NUMBER: 1998:226196 CAPLUS
DOCUMENT NUMBER: 128:292200
TITLE: ***Interleukin*** ***12***
breaks ultraviolet
light induced immunosuppression by
affecting CD8+
rather than CD4+ T cells

AUTHOR(S): Schwarz, Agatha; Grabbe,
Stephan; Mahnke, Karsten;
Riemann, Helge; Luger, Thomas A.;
Wysocka, Maria;
Trinchieri, Giorgio; Schwarz, Thomas
CORPORATE SOURCE: Ludwig Boltzmann
Institute for Cell Biology and
Immunobiology of the Skin,
Department of Dermatology,
University Munster, Munster, D-
48149, Germany

SOURCE: Journal of Investigative
Dermatology (1998), 110(3),
272-276
CODEN: JIDEAE; ISSN: 0022-202X

PUBLISHER: Blackwell Science, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Recent studies showed that injection of
interleukin (***IL***)-
12 prevents UV light mediated
suppression of contact
hypersensitivity and breaks UV-induced hapten
specific tolerance.
UV-mediated suppression can be adoptively
transferred by injecting
splenocytes from UV-irradiated mice; however,
suppression is not
transferable when donor mice are treated with
IL - ***12***
after UV-irradn. This study was performed to
elucidate the mechanisms by
which ***IL*** - ***12*** counteracts this
immunosuppression. To
characterize the cells transferring suppression,
depletion studies were
performed revealing that UV-induced suppression
is transferred via CD8+ T
cells. To investigate whether ***IL*** -
12 counteracts
UV-induced suppression by either inhibiting the
development of CD8+
suppressor T cells or inducing CD4+ effector T
cells, splenocytes from
mice, which were ***IL*** - ***12*** treated
and sensitized through
UV-exposed skin, were depleted from CD4+ T
cells and transferred into
naive mice that were subsequently sensitized.
Whereas transfer of
splenocytes from UV-irradiated mice inhibited
sensitization of recipients,
no ***inhibition*** was obsd. after
transfer of splenocytes
from UV-exposed and ***IL*** - ***12***
treated mice. Recipients
that received CD4 depleted spleen cells from UV-
exposed and ***IL*** -
12 treated donors, were still fully
sensitizable. ***IL*** -
12 also blocked transfer of UV-induced
suppression when it was

injected into UV-exposed donor animals at a time point when suppressor cells had already developed. CD4 depletion of such splenocytes did not result in a loss of the reconstitutive effect of ***IL*** - ***12***

. Thus, ***IL*** - ***12*** may break UV-induced tolerance not by inducing CD4+ effector T cells, but rather by inhibiting or inactivating suppressor T cells belonging to the CD8 subtype. REFERENCE COUNT: 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 68 OF 74 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 24
ACCESSION NUMBER: 1998:698752 CAPLUS
DOCUMENT NUMBER: 130:37230
TITLE: Increased nitric oxide (NO) production by

antigen-presenting dendritic cells is responsible for low allogeneic mixed leukocyte reaction (MLR) in primary biliary cirrhosis (PBC)

AUTHOR(S): Yamamoto, K.; Akbar, Sk. Md. Fazle; Masumoto, T.; Onji, M.

CORPORATE SOURCE: Third Department of Internal Medicine, Ehime University School of Medicine, Ehime, 791-0295, Japan

SOURCE: Clinical and Experimental Immunology (1998), 114(1), 94-101

CODEN: CEXIAL; ISSN: 0009-9104

PUBLISHER: Blackwell Science Ltd.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The levels of blastogenesis in allogeneic MLR contg. T cells from one normal volunteer and irradiated dendritic cells from 29 patients with PBC, 17 patients with chronic hepatitis type C (CH-C) and 22 allogeneic normal

controls were compared to see if there is any role of antigen-presenting

cells (APC) in the pathogenesis of PBC. The stimulatory capacity of dendritic cells from PBC was lower than that of dendritic cells from CH-C

and normal controls, which could not be attributable either to the levels of expression of surface molcs., such as HLA-DR and CD86 on dendritic cells, or to the levels of cytokines, such as IL-10 and ***IL*** -

12. Higher levels of NO were seen in the allogeneic MLR

supernatants contg. dendritic cells from PBC compared with the supernatants from cultures contg. dendritic cells from CH-C or normal controls. Moreover, dendritic cells from PBC produced 10 times more NO compared with dendritic cells from CH-C and normal controls. The addn. of NG-monomethyl-L-arginine monoacetate (L-NMMA), a known inhibitor of NO in allogeneic MLR contg. dendritic cells from PBC, resulted in a decrease of NO and increase of blastogenesis. The selective impairment of dendritic cell function, increased prodn. of NO by dendritic cells and restoration of blastogenesis using ***NO*** ***inhibitor*** in PBC have suggested a role for NO and dysfunction of dendritic cells in the pathogenesis of PBC.

REFERENCE COUNT: 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 69 OF 74 MEDLINE on STN DUPLICATE 25

ACCESSION NUMBER: 96304841 MEDLINE

DOCUMENT NUMBER: PubMed ID: 8723796

TITLE: Inhibitory effects of ***interleukin*** - ***12*** on

retroviral gene transduction into CD34 cord blood myeloid progenitors mediated by induction of tumor necrosis factor-alpha.

AUTHOR: Xiao M; Li Z H; McMahon J; Broxmeyer H E; Lu L

CORPORATE SOURCE: Department of Medicine (Hematology/Oncology), Indiana University School of Medicine, Indianapolis, USA.

CONTRACT NUMBER: R01 HL46549 (NHLBI) R01 HL54037 (NHLBI) R37 CA36464 (NCI)

+

SOURCE: Journal of hematology, (1996 Apr) 5 (2) 171-7.

Journal code: 9306048. ISSN: 1061-6128.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199610

ENTRY DATE: Entered STN: 19961022

Last Updated on STN: 19980206

Entered Medline: 19961009

AB ***Interleukin*** - ***12*** (***IL*** - ***12***), a

heterodimeric cytokine with potent biologic activity, was evaluated for effects on retroviral-mediated gene transduction into human myeloid progenitor cells in vitro. Cord blood CD34 cells were prestimulated with Steel factor (SLF), IL-3, GM-CSF, and erythropoietin (Epo) in the presence and absence of 5-80 ng/ml ***IL*** - ***12*** for 40 hr in suspension culture prior to gene transduction using viral supernatant collected from a packaging cell line containing the pLNL6 vector encoding Neo sequences. After gene transduction, cells were assayed for colony formation stimulated by Epo, GM-CSF, IL-3, and SLF, and gene transduction efficiency was determined by the percentage of G418 resistant (R) colonies and confirmed by PCR analysis. ***IL*** - ***12*** dose-dependently inhibited retroviral-mediated gene transduction into human cord blood CD34 granulocyte-macrophage (CFU-GM) and erythroid (BFU-E) progenitors. These suppressive effects could be neutralized by incubation of ***IL*** - ***12*** with polyclonal antihuman ***IL*** - ***12***. ***IL*** - ***12*** had ***no*** ***inhibitory*** effects directly on colony formation. To understand the possible mechanisms for this suppression, ELISA assays were used to detect the release of interferon (IFN)-gamma and tumor necrosis factor (TNF)-alpha, which could potentially have been induced by ***IL*** - ***12*** from CD34 cells. TNF-alpha protein release was significantly increased in CD34 cells incubated with ***IL*** - ***12***. No detectable levels of IFN-gamma were noted. Anti-TNF-alpha, but not anti-IFN-gamma, blocked the inhibitory effects of ***IL*** - ***12*** on gene transduction. Moreover, TNF-alpha, but not IFN-gamma, suppressed gene transfer to the same degree as ***IL*** - ***12***. No change of amphotropic receptor mRNA expression was noted by Northern blot analysis in cells treated with or without ***IL*** - ***12***. The results suggest that the suppressive effects of ***IL*** - ***12*** on retroviral gene transduction are, at least in part, mediated by ***IL*** - ***12*** induction of the release of TNF-alpha.

L5 ANSWER 70 OF 74 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 26
 ACCESSION NUMBER: 1996:507756 CAPLUS
 DOCUMENT NUMBER: 125:193084
 TITLE: Interferon-gamma. induced type I nitric oxide synthase activity inhibits viral replication in neurons
 AUTHOR(S): Komatsu, Takashi; Bi, Zhengbiao; Reiss, Carol S.
 CORPORATE SOURCE: Department of Biology, New York University, New York, NY, 10003, USA
 SOURCE: Journal of Neuroimmunology (1996), 68(1-2), 101-108
 CODEN: JNRIDW; ISSN: 0165-5728
 PUBLISHER: Elsevier
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 AB Type I NOS expression increases in OB neurons during VSV infection. Immunocytochem. staining of NB41A3 cells indicates constitutive expression of interferon (IFN)-gamma. receptor and type I NOS. IFN-gamma. treatment of NB41A3 cells increased NO prodn. and type I NOS-protein. In vitro replication of VSV, polio virus type 1, and Herpes Simplex virus type 1 (HSV-1) is significantly inhibited by IFN-gamma. induced type I NOS and antagonized by ***NOS*** ***inhibitors***. In contrast, while IFN-gamma. treatment inhibited influenza and Sindbis virus replication, a different pathway(s) was involved. The isoform-selective ***NOS*** ***inhibitor***, 7-nitroindazole (7NI) was used to treat mice, resulting in a 10-fold higher titer of virus in brain homogenates, and abrogated the recovery-promoting effect of ***interleukin*** - ***12*** treatment. Thus, IFN-gamma. induced type I NOS activity may play an important role in host immunity against neurotropic viral infections.

L5 ANSWER 71 OF 74 MEDLINE on STN
 ACCESSION NUMBER: 96197369 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 8625365
 TITLE: Effects of N(g)-methyl-L-arginine, an inhibitor of nitric oxide synthesis, on interleukin-2-induced capillary leakage and antitumor responses in healthy and tumor-bearing mice.
 AUTHOR: Orucevic A; Lala P K
 CORPORATE SOURCE: Department of Anatomy, University of Western Ontario,

Canada.
 SOURCE: Cancer immunology,
 immunotherapy : CII, (1996 Jan) 42 (1)
 38-46.
 Journal code: 8605732. ISSN: 0340-7004.
 PUB. COUNTRY: GERMANY: Germany,
 Federal Republic of
 DOCUMENT TYPE: Journal; Article; (JOURNAL
 ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 199606
 ENTRY DATE: Entered STN: 19960708
 Last Updated on STN: 19960708
 Entered Medline: 19960626

AB We tested whether treatment with an inhibitor of
 nitric oxide synthesis (N
 g -methyl-L-arginine, MeArg) can ameliorate
 interleukin-2(IL-2)-therapy-
 induced capillary leak syndrome in healthy or
 tumor-bearing mice without
 compromising the antitumor effects of IL-2
 therapy. Healthy or
 C3H-L5-mammary-adenocarcinoma-bearing
 C3H/HeJ mice were treated with one or
 two rounds of various doses of IL-2 (ten injections,
 i. p., every 8 h) or
 MeArg (ten injections s. c., every 8 h) or their
 combination. In an
 additional experiment, MeArg was given
 chronically in the drinking water,
 rather than s. c. to healthy mice subjected to one
 round of therapy as
 above. Mice were killed 1 h after their last IL-2
 injection to measure
 the water content of the lungs and pleural cavities
 (markers of capillary
 leakage), NO production (given by NO₂- and NO₃-
 levels in the serum and
 pleural effusion), as well as the effect of therapies
 on the primary tumor
 size and number of spontaneous lung metastatic
 nodules. Results revealed
 that all doses of IL-2 (7500-35000 Cetus
 U/injection), as well as both
 rounds of IL-2 therapy, caused capillary leakage.
 However, no pleural
 effusion was seen after the second round in any of
 the IL-2-treated
 groups. MeArg therapy, given subcutaneously (5-
 20 mg/kg(-1) injection(-1)
 in healthy and 20 mg/kg(-1) injection(-1) in tumor-
 bearing mice), did not
 ameliorate IL-2-induced capillary leakage in either
 group of mice, and did
 not compromise antitumor effects of IL-2.
 However, subcutaneous MeArg
 therapy alone reduced the growth of the primary
 tumors, the occurrence of
 lung metastases and the amount of tumor-induced
 pulmonary edema. When

MeArg therapy was given orally (1 mg/ml drinking
 water), a substantial
 drop in NO production, as well as reduction in
 capillary leakage was noted
 in IL-2-treated healthy mice. These findings
 suggest that ***NO***
 inhibitors could be a valuable adjunct to
 IL-2 therapy of cancer
 and infectious diseases.

L5 ANSWER 72 OF 74 CAPLUS COPYRIGHT
 2005 ACS on STN DUPLICATE 27
 ACCESSION NUMBER: 1995:470536 CAPLUS
 DOCUMENT NUMBER: 122:237476
 TITLE: ***Interleukin*** - ***12***
 profoundly

up-regulates the synthesis of antigen-
 specific
 complement-fixing IgG2a, IgG2b and
 IgG3 antibody
 subclasses in vivo
 AUTHOR(S): Germann, Tieno; Bongartz,
 Martina; Dlugonska, Henryka;
 Hess, Henry; Schmitt, Edgar; Kolbe,
 Ludger; Koelsch,
 Eckehart; Podlaski, Frank J.; Gately,
 Maurice K.;

Ruede, Erwin
 CORPORATE SOURCE: Institute fuer
 Immunologie, Mainz, D-55101, Germany
 SOURCE: European Journal of
 Immunology (1995), 25(3), 823-9
 CODEN: EJIMAF; ISSN: 0014-2980

PUBLISHER: VCH
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 AB The influence of the cytokine ***interleukin***
 - ***12*** (
 IL - ***12***) on humoral immune
 responses was studied in vivo.
 CBA/J mice immunized with protein antigens
 (keyhole limpet hemocyanin,
 phospholipase A2) adsorbed to aluminum
 hydroxide (Alum) develop a Th2-like
 immune response characterized by the prodn. of
 large amts. of IgG1 as well
 as some IgE but little IgG2b and IgG3 antibodies.
 IL - ***12***

is a cytokine that promotes the development and
 the activation of Th1
 cells. Th1 cells are involved in the induction of
 cellular immunity,
 which is characterized by low or absent antibody
 prodn. Some Th1-like
 immune responses are assocd. with a strong
 antibody prodn. of the IgG2a,
 IgG2b, and IgG3 subclasses. Thus, the authors
 investigated whether
 treatment with ***IL*** - ***12*** would
 down-regulate the humoral
 immune response to stimulate antibody prodn. of
 the IgG2a, IgG2b and IgG3

subclasses. The authors obsd. that administration of ***IL*** - ***12*** to mice together with protein antigens adsorbed to Alum strongly enhanced the humoral immune response by increasing the synthesis of antigen-specific antibodies of the IgG2a, IgG2b and IgG3 subclasses 10- to 1000-fold. The synthesis of IgG1 was not or only slight (2-5-fold) enhanced, whereas that of the IgE isotype was suppressed. These effects of ***IL*** - ***12*** were obsd. when high (10 .mu.g, 100 .mu.g) or low doses (0.1 .mu.g) of antigen were used for immunization. Titrm. of ***IL*** - ***12*** in vitro revealed that IgG2a is strongly up-regulated over a wide dose range of ***IL*** - ***12*** (10 to 1000 ng/day). The effects of ***IL*** - ***12*** in vivo are at least partially interferon (IFN)-gamma.-dependent because an anti-IFN-gamma. mAb in combination with ***IL*** - ***12*** prevented most of the enhanced IgG2a prodn. Mice receiving ***IL*** - ***12*** showed a strong up-regulation of IFN-gamma. but ***no*** inhibition of IL-5 synthesis by spleen cells activated ex vivo with antigen. There results suggest that ***IL*** - ***12*** is a potent adjuvant for enhancing humoral immunity to protein antigens adsorbed to Alum, primarily by inducing the synthesis of the complement-fixing IgG subclasses 2a, 2b, and 3.

L5 ANSWER 73 OF 74 CAPLUS COPYRIGHT
2005 ACS on STN DUPLICATE 28
ACCESSION NUMBER: 1995:330082 CAPLUS
DOCUMENT NUMBER: 122:103698
TITLE: Mouse ***interleukin*** - ***12*** (***IL*** - ***12***) p40 homodimer: a potent ***IL*** - ***12*** antagonist
AUTHOR(S): Gillessen, Silke; Carvajal, Daisy; Ling, Ping; Podlaski, Frank J.; Stremlo, Donna L.; Familletti, Philip C.; Gubler, Ueli; Presky, David H.; Stern, Alvin S.; Gately, Maurice K.
CORPORATE SOURCE: Dep. Inflammation/Autoimmune Dis., Hoffman-La Roche Inc., Nutley, NJ, 07110, USA
SOURCE: European Journal of Immunology (1995), 25(1), 200-6
CODEN: EJIMAF; ISSN: 0014-2980

PUBLISHER: VCH
DOCUMENT TYPE: Journal
LANGUAGE: English
AB ***Interleukin*** - ***12*** (***IL*** - ***12***) is a cytokine that has regulatory effects on T and natural killer (NK) cells and is composed of 2 disulfide-bonded subunits, p40 and p35. It was recently reported that supernatants from cultures of mouse ***IL*** - ***12*** (moIL-12) p40-transfected COS cells could inhibit ***IL*** - ***12*** -dependent responses in vitro (Mattner, F., et al., 1993). The authors have further characterized the nature of the inhibitory substance. Purified mouse p40 produced in a baculovirus expression system was found to consist of 2 species: the p40 monomer and a disulfide-linked p40 dimer [(p40)2]. The (p40)2 was 25-50-fold more active than the p40 monomer in causing specific, dose-dependent inhibition of ***IL*** - ***12*** -induced mouse Con A blast proliferation and also inhibited ***IL*** - ***12*** -induced interferon-gamma. (IFN-gamma.) secretion by mouse splenocytes and ***IL*** - ***12*** -dependent activation of mouse NK cells. Competitive binding studies on mouse Con A blasts showed that (p40)2 was equally effective as moIL-12 in competing with 125I-labeled moIL-12 ([125I]moIL-12) for binding to mouse Con A blasts. However, in contrast to moIL-12, mouse (p40)2 displayed little ability to compete with 125I-labeled human ***IL*** - ***12*** (huIL-12) for binding to high-affinity ***IL*** - ***12*** receptors (IL-12R) on human phytohemagglutinin (PHA) blasts and caused little or ***no*** inhibition of huIL-12-induced human PHA blast proliferation. Nonetheless, mouse (p40)2 was equally effective as moIL-12 in competing with [125I]huIL-12 for binding to COS cells transfected with the human IL-12R .beta. subunit and expressing low-affinity ***IL*** - ***12*** binding sites. Thus, (1) the majority of the structural determinants required for binding of ***IL*** - ***12*** to its receptor are contained within the p40 subunit, but p35 is required for signaling, (2) the p40 subunit of ***IL*** - ***12*** interacts with the .beta.

subunit of IL-12R, and (3) (p40)2 may be a suitable
IL -

12 antagonist for studying the role of
IL - ***12***

in various immune responses in vivo as well as in
vitro.

L5 ANSWER 74 OF 74 CAPLUS COPYRIGHT
2005 ACS on STN DUPLICATE 29

ACCESSION NUMBER: 1994:189371 CAPLUS

DOCUMENT NUMBER: 120:189371

TITLE: Differential effects of

interleukin -

12 on the development of
naive mouse CD4+ T
cells

AUTHOR(S): Schmitt, Edgar; Hoehn, Petra;
Germann, Tieno; Ruede,
Erwin

CORPORATE SOURCE: Inst. Immunol., Mainz,
D-55101, Germany

SOURCE: European Journal of
Immunology (1994), 24(2), 343-7

CODEN: EJIMAF; ISSN: 0014-2980

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The influence of interleukin (***IL***)-

12 and IL-4 on the

differentiation of naive CD4+ T cells was studied
in an accessory

cell-free in vitro system. Dense CD4+ T cells were
purified from

unimmunized mice and activated using

immobilized anti-CD3 monoclonal

antibodies (mAb) in the presence of IL-4,

IL - ***12*** , or a

combination of both cytokines, and restimulated
after 6 days by

re-exposure to anti-CD3-coated culture wells. T
cells initially activated

in the presence of IL-4 produced substantial amts.
of IL-4 and trace amts.

of interferon (IFN)-.gamma. after restimulation at
day 6 with plate-bound

anti-CD3 mAb. By contrast, T cells primed in the
presence of ***IL***

- ***12*** produced high levels of IFN-.gamma.
and only minimal amts.

of IL-4, thus indicating that IL-2 and IL-4 by acting
directly on

stimulated naive CD4+ T cells support the
development of TH1 and TH2

cells, resp. When naive CD4+ T cells were
stimulated in the presence of

IL - ***12*** together with IL-4 in
comparable concns., the

effect of ***IL*** - ***12*** on TH1
differentiation was largely

inhibited by IL-4. ***IL*** - ***12***
exerted ***no***

inhibitory effect on IL-4-induced TH2
differentiation but rather

enhanced the prodn. of IL-4 after restimulation of
the resp. T cells.

Decreasing amts. of IL-4 in combination with a
high level of ***IL*** -

12 led to an increasing prodn. of IFN-
.gamma. by the emerging T

cells and, simultaneously, to a relatively high
prodn. of IL-4. These

data were confirmed by time-course expts. which
revealed that the delayed

addn. of IL-4 to ***IL*** - ***12*** -primed
T cell cultures resulted

in a gradual restoration of IFN-.gamma. prodn.
whereas in parallel the

secretion of IL-4 was not reduced over a wide
period of delay (6-72 h).

These results, therefore, demonstrate that (a) IL-4
dominates the effect

of ***IL*** - ***12*** , (b) ***IL*** -
12 promotes the

development of TH1 cells; however, in the
presence of ***IL*** -

12 and relatively high levels of IL-4 also
the development of

TH2-like cells is slightly enhanced by ***IL*** -
12 , and (c)

high amts. of ***IL*** - ***12*** in
combination with relatively low

levels of IL-4 give rise to a T cell population that
upon rechallenge

exhibited a cytokine profile resembling that of TH0
cells.

=> log y

COST IN U.S. DOLLARS

SINCE

FILE TOTAL

ENTRY SESSION